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Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER

INCORPORATING

THE INDEX OF OTOLARYNGOLOGY

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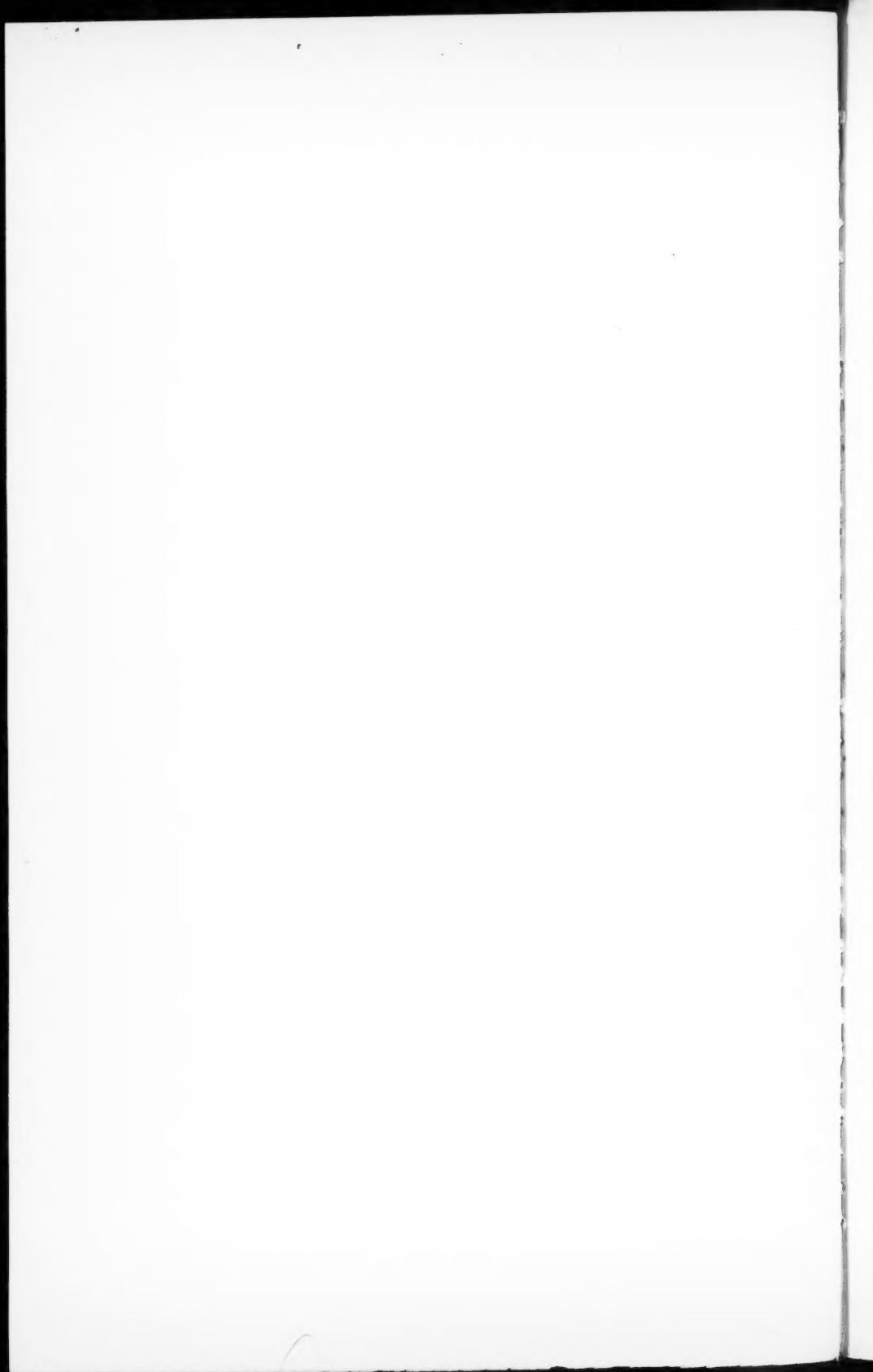
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Samuel H. Loeb,





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VOL. XXXVII.

MARCH, 1928.

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I.

HANAU W. LOEB—A TRIBUTE OF FRIENDSHIP.

By DR. LEON HARRISON,

ST. LOUIS.

It is difficult to draw a pen-picture of a man as many-sided as my lifelong friend, Dr. Hanau W. Loeb. No set list of virtues possessed by such a man presents him to view; any more than an actual photograph of his physical semblance could reveal his mental grasp, his driving power, his endearing human qualities. I may begin by saying that no man known to me in this great city has left such an impact on its collective life as Dr. Loeb. No one man has in so many ways built up the institutional life of this community. He organized a great hospital service, developed a remarkable professional career, edited famous medical journals; and was the executive and creative head of an important medical school. He composed standard works in his field of medicine. He was honored by the Government for his brilliant medical services during the war. And in the "War without Discharge," between life and death, between poverty and disease, Dr. Loeb was a gallant combatant, offering up time, effort and patient research in abundant measure to those that could give in return nothing but a word of thanks, a warm handclasp, or the tears that dimmed the eyes of those too moved to find words of gratitude. To this high

cause of Humanity above all, Dr. Loeb finally sacrificed his splendid life. For such a man as he did not, as men think, die in a moment, of a sudden seizure; nay, he died inch by inch, year by year, burning up his marvelous vitality in a strenuous, manifold career of public and humanitarian service in this city, to which I do not know a parallel.

For this one man did the work of half a dozen men; even between his treatments of patients in his office, he stole moments for the dictation of his medical works. He was an author, an editor, a great practitioner of medicine, a professor and teacher of his specialty; the organizer and executive of a great School of Medicine; and the Chief of Staff of a wonderful metropolitan hospital. He was all that and more. He assisted vigorously every great philanthropic cause in the city of St. Louis. He delighted in social life, and injected into every friendly gathering the same enthusiasm, the same geniality, the same sheer joy of living. And yet he never seemed tired. He was unhurried, apparently free from care; the constant solitudes within were never evidenced in a frowning brow, in physical irritability or in apparent fatigue. He was made seemingly to carry great and varied burdens easily, and to esteem them privileges. He could forget all his responsibilities in a jiffy, and join in a game of cards or in his favorite recreation of golf with the abandon of a schoolboy.

And with all his countless preoccupations, he always had time, much time, for his friends. Dr. Loeb had a genius for friendship. Years ago, when the cares of life were not as insistent with the present writer and with Dr. Loeb, it was my privilege and joy to be his constant companion. We were inseparable friends. We met at luncheon every day, with Dr. Jacob Friedman, of blessed memory; and frequently with that brilliant editor, William Marion Reedy, and the late George Tansey; and jest and repartee and words of wit and wisdom were tossed from man to man, so that many a little informal luncheon of this kind was a veritable symposium; and where Dr. Loeb sat was the head of the table. Almost imperceptibly he guided conversation, he drew this man out and then the other. His delightful geniality and social charm seemed to cement the company together. And many a worthy project

in the interest of some struggling student, some forlorn hope or some much-needed philanthropy sprang from these pleasant luncheons.

It was Dr. Loeb's personal magnetism, his compelling enthusiasm, that drew people to him, and made it easy for them to follow him in any good cause. He didn't have to explain, or argue, or persuade, though he could do all these convincingly well. He just had to lead, and men followed him, as we follow those whom we heartily like—without question, without a moment's doubt. These lovable qualities in Dr. Loeb that made people like him so much explain a great deal of his leadership, in addition, of course, to his fine intellectual and moral qualities. For other men had brains and character, yet they could not begin to command any such following, or to rivet friends to themselves as staunchly and steadfastly as Hanau Loeb.

I remember when Israel Zangwill, the brilliant Jewish novelist of England, visited St. Louis, Dr. Loeb and I went down to meet him on his arrival; and both of us, being at the young hero-worshipping age, were constantly in the company of this sparkling mind, that gave forth epigrams and witticisms with as little effort as average men make in emitting wearisome commonplaces. Dr. Loeb worshiped intellect; and he arranged many luncheons and entertainments for Zangwill during his sojourn here; and he drew him out during the social hour that followed the demi-tasse, when men lean their elbows on the table and pull their chairs a little closer together, mellowed by food and wine and social contact; and are eager to hear what tidings this messenger of the Muses brings from far-off lands. And never will I forget one such occasion, when a company of kindred spirits foregathered with Israel Zangwill and Dr. Loeb at the Noonday Club, around the mahogany. William Marion Reedy was there; Fred Lehman, then the Solicitor-General of the United States; Isaac Lionberger, an under-Secretary in the Cabinet; and Mr. Ben Altheimer, the giver of the feast. For many hours we sat there when the luncheon was over, until almost evening; and never did I hear such wonderful talk, flavored with Attic salt, playful, sparkling with sudden sallies of wit; and deepening into serious com-

ments on society and literature. It reminded one of the famous Symposium at which Plato was a guest in Old Athens, and his teacher Socrates and their boon companions; when wit flashed, and humor smiled, and mellow wisdom flowed abundantly. On this occasion, as on many similar ones, I well remember the roguish smile of Dr. Loeb, his provocative remarks that drew fire all around the merry circle; and the evident appreciation and sympathetic spirit that made this company of congenial spirits address their many sallies almost involuntarily to him.

I like to remember my old friend and companion in such scenes as this.

And the qualities that made him beloved there and everywhere, may I not say, were legitimately his. They came to him from the dear, devoted mother, and the splendid old father with a beautiful head like Pasteur's, in the old family home in Columbia, Missouri. Many a glad and many a sad day we shared together there. And Dr. Loeb's pride and joy it was to see the fine fruits of his brotherly solicitude in the remarkable career and professional distinction of his brother, Dean Isidor Loeb, and in the fine manhood and medical eminence of his brothers Clarence and Virgil. Indeed, I can still see him in happy mood at the wedding of his dearly beloved sister, when the table rocked with merriment, and from the beaming countenance of Hanau shone forth a happiness that sprang from all the joyful family years of Auld Lang Syne.

I would like to say much of the unstinted generosity of my old friend, of how lavish he was with his resources, even when they were scanty, to those he loved, and for the great things that he cared for. But no man ever allowed less to be known of his private benefactions than Dr. Loeb. I know, however, how great his sacrifices were, when inspired by affection and a sense of duty. Even in the days when he had but little, he would divide his last dollar with one in need who appealed to his generous heart.

But I would rather remember what a man was than what he did. A virile, expansive personality like Dr. Loeb's was never fully expressed in the achievements of his life. He was a big-hearted, affectionate, lovable man. And wherever he

lived and moved he sounded a note of sanity, of sweet reasonableness, of a happy nature that smiled its way through life. I can see him at home with his lovely and devoted wife, playing on the floor with his baby girl. I can imagine myself once more climbing the Alpine passes with him, and with Dr. Max Myer, and Judge Irwin Barth, in our early salad days; swinging along from dawn until dusk, uphill and downdale, all of us happy as the day was long; and always Dr. Loeb breathed his own gay, whimsical spirit into all of the little company.

And I can see him when the strong machinery of his body, alas, suddenly broke down, and after many weary weeks of pain and danger, he could once more be seen for a little while by a few old friends. He knew his own condition. He was aware of the fact that he had reached the end of his tether; but he looked steadily into the darkness, unafraid. He never flinched, he never weakened. His body was broken, but his spirit remained indomitable. "I am satisfied, Doctor," he said to me, "indeed, I am thankful for sixty good years of happiness and usefulness." He realized that he had lived, and lived abundantly; he had worked and worked fruitfully; he had built not for a day but for all time. If his time was up—and he felt that it was—he was content to go. For his work was done, and well done. He had fought the good fight. He had borne the burden and the heat of the day; and though the shadows had gathered around him sooner than he had hoped, yet he knew at that awful gripping moment that life is measured less by its length than by its contents. And so he waited for his earthly end, only postponed for a few short months, without fear. He lived to see the dedication of the new Jewish Hospital, for which he had toiled and striven greatly. He lived to speak a few moving words on the happy day when its hospitable doors were flung open to receive the stricken, the sick and the helpless. Although a Jew, and a faithful Jew, loyal in every instinct of his being to his people and his Faith, as well as to the whole brotherhood of man, a Novena, a nine-day devotional service, was held for him by the Catholic Church, and prayers for his restoration to health and life were fervently uttered in his behalf by the Catholic Sisters that he had befriended, and whose poor he had tended and cared for. For this man had served them all, Catholic and Protestant,

as well as the needy of his own race and people. He knew no barriers of race or creed. He gave abundantly of his prodigious energies, and of his unusual talents, for the service of humanity.

Such a life needs no memorial-stone on whose cold surface his virtues shall be carved. For his works are his living memorial. The great hospital that he toiled for shall perpetuate his name, in the loving and grateful memory of countless men and women. The famous school of medicine, to which he gave up his manhood's span of life, will long recall his creative genius. Many hearts will bless him—many lives that he brightened and cheered.

I am but one of a great multitude who tenderly remember this true and faithful friend of long ago. It is needless to praise when a true description is so high a eulogy. Deep in our hearts we will carry the precious memory of this high-minded gentleman, the sacred memory of one who greatly helped and nobly served his fellowmen.

II.

HANAU W. LOEB: A BIOGRAPHICAL SKETCH.

By W. E. SAUER, M. D.,

ST. LOUIS.

Dr. Hanau Wolf Loeb was born in Philadelphia in 1866. After his early education in the East, he attended the University of Missouri, taking his A. B. degree from that institution in 1883 and an A. M. in 1886. While there he was awarded membership in the honorary fraternity Phi Beta Kappa. He then began his medical studies in the Ensworth Medical College at St. Joseph, Mo., which were completed in the College of Physicians and Surgeons, then a part of Columbia University in New York, in 1888. After practicing medicine in St. Joseph, Missouri, for two years he moved to St. Louis in 1890, where, with a number of colleagues, he organized the Marion Sims Medical College. He continued with this school as head of the Department of Otolaryngology and secretary of the Faculty until its consolidation with the Beaumont Medical College. He served as secretary of the Faculty of the combined schools until they were taken over as the Medical Department of the St. Louis University. In 1913 he was made Dean of the Faculty of the School of Medicine of St. Louis University. It was in this capacity that he demonstrated his remarkable leadership and executive ability. Dr. Loeb had unusual capacity for work, and his uncanny power of overcoming seemingly insurmountable obstacles won the admiration of all his co-workers. He was ambitious to place the Medical Department of the St. Louis University among the foremost institutions of its kind in this country. The results accomplished were largely due to his untiring efforts, often at a personal sacrifice. His ideals were ever of the highest order, and neither personal gain nor at times the sacrifice of personal friends, affected his decisions when the advancement of the school was at stake.

In 1905, Dr. Loeb was made president of the American Academy of Ophthalmology and Otolaryngology. At this time

the membership of this society was composed largely of men from the West or Midwest, and it was chiefly through his efforts that its scope was widened until it became national in character. In 1917, he became chairman of the Section of Otolaryngology of the American Medical Association. In 1924, he was honored with chairmanship of the American Laryngological, Otological and Rhinological Society. He was a fellow of the American Laryngological Association, the American Otological Society and the American College of Surgeons. He was a very active member in all these organizations and served on the councils of several of them for many years. During the early years of his professional life he was active in the local and state medical organizations, always working to place medicine on a higher plane.

Dr. Loeb was among the first to offer his services during the World War and was appointed Major in 1917. He wrote a handbook on "Military Surgery of the Ear, Nose and Throat," and was also actively engaged in organizing the medical forces in his home city. He became a Colonel in the Medical Reserve Corps at the close of the war.

Dr. Loeb made many contributions to Otolaryngology, the best known of which are his researches on "The Nasal Sinuses and the Determination of Their Capacity, Area, Position and Relations," and a textbook on operative surgery of the ear, nose and throat. He was editor of the ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY. Dr. Loeb was one of the organizers of the American Board of Otolaryngology and was secretary of that body from its organization until his death, July 6, 1927.

He was one of the most active workers for the advancement of the St. Louis Jewish Hospital, and it was largely through his efforts that its present new building became a reality. He was Chief of Staff of this hospital during the last years of his life.

His name appears, together with those of two of his brothers, in "Who's Who in America."

One of Dr. Loeb's most admirable traits was his unselfish friendship for, and untiring efforts in the advancement of his younger competitors in the otolaryngologic field. He was ever at their service and his advice was always freely given.

His capacity for detail was prodigious. Few men have been privileged to be chosen for so many important posts, and few, indeed, could have filled so great a number of them as creditably as he. He was a man of varied interests and he excelled in all of them.

It is true that he had more things in his mind at times than most human brains could carry. However, nothing was ever left unattended if it was placed before him as a written document. No written request was ever neglected nor was any written promise forgotten.

He was first, last and always an idealist of the purest ray serene, and there was nothing materially selfish about his idealism. His life was devoted to the betterment of something or somebody worth while.

III.

A STUDY OF THE LOWER END OF THE
ESOPHAGUS.*

BY HARRIS P. MOSHER, M. D.,

BOSTON,

AND

G. W. MCGREGOR, M. B.,

TORONTO.

For the past two or three years there has been a great revival of interest in cardiospasm. The English laryngologists have been especially active in discussing the condition. This revival of interest is due to the fortunate fact that a number of cases of cardiospasm have come to autopsy, and a fundamental observation resulted from the study of the specimens, namely, that the nerve control of the musculature of the esophageal wall, located between the two chief muscular layers in groups of ganglion cells known as Auerbach's plexus, is diminished or in places abolished, due to pathologic changes which have destroyed the ganglion cells. Stokes was the first to make this observation. Others have confirmed it, notably Kelley and Hurst. This observation is a very welcome addition to our previous scanty knowledge of the microscopic pathology of the esophagus.

Subject.—The title of the paper is "A Study of the Lower End of the Esophagus." The first part deals with the histology of the normal esophagus from infancy to 61 years of age. It deals also with the microscopic findings in my one autopsy specimen from a case of cardiospasm. The second part is clinical, and deals with a series of nine cases of cardiospasm

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The X-rays illustrating this article were taken by Dr. A. S. Macmillan, roentgenologist of the Massachusetts Eye and Ear Infirmary.

in which the diagnostic barium bougie was used and in which dilatation of the lower end of the esophagus was carried out under sight.

Material—

Fetus, five months.....	1
Microscopic sections.	
Infants dead at birth.....	3
Hardened in formalin and anterior wall of esophagus and stomach removed.	
Infants dead at birth.....	2
Microscopic sections of esophagus.	
Infant, one year.....	1
Microscopic sections.	
Child, two years.....	1
Microscopic sections.	
Child, twelve years.....	1
Microscopic sections.	
Adults, 40-61 years.....	5
Microscopic sections.	14

These adult specimens included a marked case of arteriosclerosis; a case which died of cardiorenal disease; a case of stricture of the esophagus which died of septic pleurisy and peritonitis after perforation of the esophagus by a bougie; a case of cardiospasm of fifteen years' duration which died of tuberculosis of the lung.

Conclusions.—In the gross and microscopic examination of fourteen specimens of the human esophagus, varying in ages from birth to 61 years, neither the writer nor Dr. McGregor, who has done the microscopic work for this paper—he has examined 1,500 sections—have been able to find an anatomic sphincter at the cardiac end.

Physiologic experiments have shown that the musculature of the lower end of the esophagus or of the upper end of the stomach acts as a sphincter. In the resting state the terminal portion of the esophagus is practically closed. This is due to the presence of deep vertical folds and much elastic tissue. In

the esophagus the elastic tissue is most abundant at the lower end. The natural twist of the terminal portion of the esophagus helps to maintain the closure. The pressure of the left crus from behind and of the liver from the front aid in the same result. In a typical case of cardiospasm the obstruction at the lower end of the esophagus is slight. It is often accompanied by a backward bend of the terminal portion. An instance of this backward bend has been found by the writer at birth. It was associated with an upward swing of the stomach so that its long axis was horizontal. This change in the position of the stomach may be a causative factor in the deformity of the esophagus.

In nine consecutive cases of cardiospasm the diagnostic barium bougie has shown a waistlike constriction of large caliber—19 to 20 mm.—at the terminal portion of the esophagus above the level of the crura. In all but two of the cases the narrowing dilated under light pressure (three or four pounds) until the esophagus reached its normal width—31 mm.

The waistlike constriction takes the form of the lung tips and is most pronounced when they are inflated.

The Sippy rubber dilator, painted with stripes of barium and rubber cement, enables the operator to dilate the lower end of esophagus under sight.

The observation made by Stokes that in cases of cardiospasm the ganglion cells of Auerbach's plexus are much diminished and in places practically wanting was found to hold in the one case of the writer's which came to autopsy. This esophagus was sectioned from above downward at seven different levels, and 345 complete sections were examined and the ganglion cells in Auerbach's plexus counted. In the narrow terminal portion of the esophagus no ganglion cells were present in 100 sections.

The chief observations made of late years by physiologists working upon the esophagus are as follows:

The Nerve Supply of the Esophagus.—Contradictory results have been obtained experimentally in animals after cutting the vagus nerve of both sides. A recent research article states that after section of both vagus nerves the lower half of the esophagus—the part supplied by the involuntary muscle—regains its tone in twenty-four hours, whereas the upper half does not

show peristalsis again for three months, and then only feebly. This observer maintains that cutting the sympathetic nerves has no added effect. In these experiments there was no dilatation of the esophagus. Another observer, whose experiments were carried out some years ago, holds that after section of the vagi the esophagus does dilate. It would seem to me that it should. The motor cells in Auerbach's plexuses are out of commission after section of the vagi unless there is a round-about nerve supply of which we do not know.

The Cardiac Sphincter.—It will save time to admit that the musculature of the lower end of the esophagus or of the upper end of the stomach has a weak sphincter action, regardless of the presence or absence of an anatomic sphincter. Not to admit this would do violence to certain findings in experimental physiology. Cannon made the observation that when the stomach is filled with a neutral solution it runs back and forth between the esophagus and stomach without hindrance. When, however, the solution is made acid it is shut off from the esophagus and retained in the stomach. He believes, therefore, that there is an acid control of the cardiac sphincter, the nerve channel being through the cardiac plexus.

I have never been able to make out an anatomic sphincter at the cardiac end of the esophagus. In the fourteen specimens ranging in age from birth to 61 years no definite sphincter appears. A long time ago Hyrttil described an incomplete sphincter. Recently Hurst figured a very definite one at birth. My observations so far have not corroborated his. In one infant examined by sections there is a slight increase in the mass of the circular fibers at the lower end of the esophagus, but similar slight increases are found in the same specimen higher up. (See Figs. 1-5.)

We are indebted to Cannon for another important observation. He found experimentally that pressure enemias of a given portion of the intestinal tract produced experimentally was followed—four hours—by destruction of Auerbach's plexus and a cessation of peristalsis. This links up with the observation of Stokes and the later English pathologists that in cases of cardiospasm there is a loss or diminution of ganglion cells in Auerbach's plexus.

The Muscularis Mucosa.—The muscularis mucosa is thickest at birth, and thickest also at the lower end of the esophagus. From birth to adult life it increases in size only one-tenth, whereas the two outer muscular coats of the adult esophagus have increased in size to three times their measurement at birth. (McGregor.) (See Fig. 6.)

The action of the muscularis mucosa I find hard to understand. It is a longitudinal muscle and is placed a little below the epithelium. It winds in and out of the folds of the mucous membrane and is thickest where they are deepest, namely, at the terminal portion of the esophagus. This at first seems suggestive, but the axis of the folds is vertical, the same as the axis of the muscle. The muscularis mucosa, therefore, runs the wrong way to cause them. It probably reinforces the action of the longitudinal coat, the outer of the two main muscular coats of the esophagus.

The Elastic Tissue of the Esophagus.—The arteries of the body contain a large amount of elastic tissue, the presence of which brings any given vessel, for instance, the descending aorta, back to its normal size after a wave of blood has passed. The esophagus also contains elastic tissue. It is most abundant in the submucosa, although a certain amount is scattered between the bundles of the two main muscles. The elastic tissue is greatest in amount at the lower end of the esophagus. This is true of the infant as well as of the adult. Even in the disorganized esophagus of my one autopsy specimen from a case of cardiospasm the elastic tissue was more abundant at the lower end. The dog, in spite of having control of the whole length of his esophagus through voluntary muscular fibers, has more elastic tissue at the lower end than at the upper. Examination shows that the elastic tissue is scanty in the intestines. (See Figs. 7-9.)

The Comparative Anatomy of the Esophagus.—This is a rather ambitious heading, because I have only a little barnyard comparative anatomy to give—that is, a description of the esophagus of the dog, the cat and the rabbit. The dog seems to stand between the cat and the rabbit, as far as its esophagus is concerned. The muscles are voluntary throughout, and are very strong. The most striking thing about the dog's esophagus is the tremendous supply of mucous glands. The dog's esopha-

gus, in consequence, must be a very greasy affair. It needs to be, considering the kind of food which he eats and his manner of eating. The characteristic ease with which a dog can vomit is due to the voluntary muscular fibers, helped out by the abundant lubrication furnished by the excessive gland supply. (See Figs. 10-12.)

In the rabbit the position of the muscular coats is reversed. The circular coat is on the outside and the longitudinal on the inside. The rabbit is said to have a cardiac sphincter. There is a slight thickening and the muscular layers interdigitate at the lower end, but I was disappointed in not finding a frank sphincter. The rabbit has few glands in the esophagus.

The Cat.—The esophagus of the cat is more like the human. It has both voluntary and involuntary muscular fibers. It has almost no glands. That the cat should be like the human is not surprising, because there are many human cats, especially female.

In the three animals just mentioned the muscular layers often run diagonally and interdigitate. In man the layers are truly circular or longitudinal.

Muscularis Mucosa.—In the esophagus of the dog there is a thick subepithelial layer of stout connective tissue. In this a muscularis mucosa of fair size is buried. No such extensive layer of subepithelial connective tissue is found in man, in the cat or in the rabbit.

The muscularis mucosa is very large in the cat, and thin in a rabbit. In the three animals under discussion the muscularis mucosa, the same as in man, is thickest at the lower end of the esophagus.

The Resting Form of the Lower End of the Esophagus.—Even if there is no anatomic sphincter at the cardia the esophagus gets along well without one. The lower end seems normally to be closed. This is brought about by the action of the elastic tissue and by the folding together of the deep vertical pleats characteristic on this part of the esophagus. In addition there is the natural twist of the esophagus on itself from right to left, and the pressure of the liver in front and the projection of the left crus into the esophagus from behind. With each inspiration the inflated lung tips press on the esophagus and momentarily close it. Keith describes the esophagus as one

long sphincter. Peristalsis is a sort of traveling sphincter. (See Figs. 13-22.)

THE PATHOLOGIC CHANGES FOUND IN CASES OF CARDIOSPASM.

The pathology which produces the main symptom of which the patient complains in cardiospasm—namely, starvation—is found in the terminal portion of the esophagus. It consists of a narrowing helped out in advanced cases by a backward bend. At the present time the English laryngologists are very active in investigating this subject. The English observers have been very fortunate in having a large number of autopsy specimens from cases of cardiospasm. For instance, Kelly, in a recent and very beautiful paper, figures eight cases collected by Owen Moore and adds three or four of his own. (See Figs. 23-27.)

A number of years ago Stokes brought out a new pathologic finding in cases of cardiospasm. He found in three esophaguses from patients who at their death suffered from cardiospasm that the motor ganglion cells which run the musculature of the esophagus and which are grouped in nerve ganglia known as Auerbach's plexuses were diminished in number. This observation was new and fundamental. When you think it over, the observation is what you would expect. When a muscle does not work, either the muscle fibers are faulty or their nerve supply is wrong. Since the publication of this observation other writers have used it as the starting point of the condition which they believe to be the cause of cardiospasm. Notably among these is Hurst. Last year, in an article in the *Lancet*, he mentioned that cardiospasm was caused by the tonic contraction of the cardiac sphincter due to the fact that fibrosis and infection of Auerbach's plexus had destroyed the motor ganglion cells. When Auerbach's plexus is normal, at the proper time in swallowing it sends the required stimulus to the cardiac sphincter, causing this to relax and allow food to pass. On the other hand, when the plexus is diseased, the sphincter remains tonically contracted until the weight of the food—a column eight inches high is usually sufficient—in the esophagus tires out the sphincter. All exponents of the cardiac sphincter admit that it is a weak affair.

The Findings in the Writer's Case of Cardiospasm.—The writer has had but one autopsy specimen from a case of cardio-

spasm. For him it is really a lone star case. The patient was a man in the fifties who had suffered from cardiospasm for fifteen years. He had been through the typical treatment of dilatation with bougies, under ether and without, at the hands of his physicians and at his own hands. He would go along months or even a few years with relief and then would shut down. He was a stone cutter and finally died of tuberculosis.

His X-ray showed a much dilated esophagus, which sagged markedly to the right. It was very narrow at the lower end, and there was a pronounced backward bend at this point. The esophagus, when removed from the body, allowed water to pass into the stomach slowly and in small amounts. On pouring in water the esophagus filled to the top, ballooned out and sagged to the right, reproducing the X-ray picture. (See Figs. 23-35.)

On gross examination the dilated part of the esophagus was found to be twice as thick as the normal. Both muscular coats were of equal thickness, measuring together at their thickest portion 2.8. The thinnest portion was at the lower end, where it measured 1.9. Microscopic examination showed that both muscular layers were thickened equally. It has been stated that only the circular muscular coat was hypertrophied in these cases. By comparison with the dilated portion, the narrow terminal part looked at first glance as if it was thinner, even atrophied. On close examination this was not the case. It was of normal thickness.

Dr. McGregor has made a very exhaustive microscopic examination of this specimen.

The first finding noted was an increase of connective tissue and the round cell infiltration, and both were confined to the superficial layers of the esophagus, the mucosa and submucosa. The second observation was that the blood vessels were much increased in number and size. Both arteries and veins shared in the enlargement alike, and the enlargement suggested arteriosclerosis.

Contrary to the statement of Kelly that Auerbach's plexus is found only where involuntary muscle fiber exists—that is, from the middle of the esophagus to the end and is not found at the top—this specimen shows that the plexus is

found in small numbers at the top among the voluntary muscular fibers. (See Figs. 36-38.)

Meisner's plexus, which runs the muscularis mucosa, is an offshoot of Auerbach's plexus. As it is smooth muscle and exists throughout the esophagus, it must have a motor nerve supply. Stated broadly, however, the statement is true that Auerbach's plexus is found most abundantly at the lower part of the esophagus, where the involuntary muscular fibers predominate. Even in the dog's esophagus, where the musculature is voluntary throughout, Auerbach's plexus is found at the top.

The most important observation has to do with the number of ganglion cells in Auerbach's plexus at various levels. Sections were taken from above downward at seven different places. Auerbach's plexus was found in scanty numbers at the top of the esophagus. In the terminal portion no ganglion cells were found in one hundred sections. Fibrosis of the ganglia was not found (as reported by Hurst). The changes noted consisted of an infiltration with plasma cells in the submucosa and a disappearance of the ganglion cells. These observations, though differing somewhat in detail from the observations of Kelly and Hurst, confirm the findings of these two men and of Stokes, who made the original observation, which was to the effect that in the esophagus in cases of cardiospasm the ganglion cells in Auerbach's plexus are diminished, especially at the terminal portion.

Having found a striking increase in the number and size of the blood vessels in my cardiospasm case, I was fortunate in having the esophagus of a man of 61, who died of pneumonia after the first stage of a laryngectomy, and who showed at autopsy general arteriosclerosis of a most advanced type. His esophagus on microscopic examination showed the same condition of the blood vessels as was found in the case of cardiospasm, but what was of even more interest was the fact that the ganglion cells of Auerbach's plexus were decidedly diminished in number (McGregor). In other words, the esophagus seems to share in the general systemic disturbances which cause widespread degeneration of the organs of the body as a whole. I had not been accustomed to think of the esophagus in this light.

The observation that the ganglion cells of Auerbach's plexus are diminished or practically wanting at the terminal portion of the esophagus in long standing cases of cardiospasm is most important. But I do not think that it is the whole story. Something must keep the terminal portion of the esophagus closed when its musculature has ceased to function on account of the loss of motor nerve supply. Three things might do this, namely, pressure from the outside, the action of the elastic tissue, bend or twist. In my opinion, all three are factors. Of the three I think that bend or twist the most important.

In a typical case of cardiospasm the dilated upper portion of the esophagus is succeeded by a short narrow terminal portion. This often has a backward bend. It was conspicuous in my case. An instance of this backward bend, associated with an enlargement and an S-shaped curve of the esophagus above, has been found by the writer even at birth. In this baby the stomach had swung upward so that its long axis was horizontal. This change in the position of the stomach I believe to be a causative factor in causing the deformity of the esophagus. (See Figs. 39-41.)

While on this subject of the backward bend of the terminal portion of the esophagus, I might add that an eleventh hour bit of preparation for this paper was the gross examination and sectioning of the esophagus of a five months' fetus. On opening the chest and abdomen and exposing the esophagus for removal, it was found that the terminal portion made a U-shaped bend down and up round the left lung tip, so that the tip lay between the fundus of the stomach and the esophagus. My conjectural explanation is that the bend was due to a disproportionate growth between the lungs and the esophagus. Whether this is the right explanation or not, the fact remains, as my specimens show, that the stage may be set for trouble with the esophagus, not only at birth but long before.

The Findings in a Five Months' Fetus.—On gross examination the lower end of the esophagus, as just stated, was seen to make a U-shaped turn round the left lung tip. The upper part of the esophagus was much thinner than the lower half.

The examination of microscopic sections showed that the thinness of the upper half of the esophagus was due to the fact that the circular fibers were very sparse at the top. The

muscularis mucosa was present throughout the whole length of the esophagus. It was thin at the top and at the bottom, but relatively very thick at the middle of the esophagus. There was no evidence of an anatomic sphincter at the cardiac end.

Auerbach's plexus was found throughout the whole length of the esophagus. The number of ganglion cells to a section averaged 27, practically the same as in the infant.

THE DIAGNOSTIC BARIUM BOUGIE.

The diagnostic bougie has given me a visualization of the lower end of the esophagus which I have never had before. It has given me information which neither the X-ray nor the esophagoscope have furnished and has made me doubt some of my observations, made with the esophagoscope, at the lower end of the esophagus in years past. I have always been partial to large examining tubes. If the large tube will lead the observer astray, a small tube is a much greater sinner in this respect. The matter may be summed up as follows: It is difficult to be sure of what you see at the end of a twenty-one inch tube, large or small, and it is equally hard not to see what your mind has been made up to see. One observer sees spasm of the esophageal muscles, another spasm of the crura, another spasm of the cardiac sphincter, and finally another observer always sees stricture. (See Figs. 42-44.)

In using the diagnostic bougie, the first thing was to establish the normal dimensions of the lower end of the esophagus in its various diameters. This was accomplished by passing the bag on "Bill," the accomplished giver of enemata on the private ward of the infirmary. He was the only volunteer and will live long in my grateful memory.

At first the barium bag was used for diagnostic purposes only. It was soon found, however, that even the pressure exerted by the bag was sufficient to cause some dilatation. Then the Sippy bag, as it was stronger, with barium and cement lines, was used both for purposes of diagnosis and dilatation. Owing to the silk cover, it is possible to exert a pressure of ten to fifteen pounds. So far I have not gone beyond four pounds. I admit that watching the esophagus dilate under direct vision, which the fluoroscope allows one to do, makes one a bit timid. You feel as if the esophagus may burst at any moment.

I have had one fatality, which may have been traceable to the bag. The dilatation was without incident. Two weeks later the patient became violently ill and died with symptoms of septicemia. An abdominal exploration for the purpose of finding a rupture of the esophagus was negative. The only pathologic condition discovered was an enormously large liver, which previous X-rays showed the patient had had for some months. A small section taken from this at the operation showed a violent infection, the organism being streptococcus hemolyticus. Puncture of the liver at the time of the exploratory operation for abscess was negative. No autopsy could be obtained and so the exact condition of affairs will never be known. I am never entirely happy in passing any instrument through the esophagus, because I have had my full share of tragedies, even from the simple passing of an elastic bougie.

Findings With the Barium Bougie.—The ordinary barium picture of the esophagus in cases of cardiospasm is familiar to us all. The terminal portion comes to an awl-like point, and this point is continued as a thin ribbon of barium for an inch or an inch and a half, and then broadens out on the anterior wall of the stomach. Watched through the fluoroscope, you see the esophagus end in the same awl-like point, but at each inspiration, as the lung tips fill, the tip of the point seems to be amputated for half an inch or more. This happens regularly with each inspiration. From this picture it is hard not to draw the conclusion that the esophagus is strictured to a small caliber. On passing the diagnostic bag, however, the picture changes. After halting a second at the lower end of the esophagus the bag passes into the stomach as if it slid over a slight obstruction. On inflating the bag, the seemingly slight stricture disappears, and in its place a waistlike constriction appears at the level of the dome of the left half of the diaphragm. Under light pressure—three or four pounds—on the second or third passage of the bag, the esophagus can be seen to dilate to normal caliber. This dilatation is followed by a great improvement in the symptoms for weeks or months.

In my series of nine recent cases of cardiospasm, this waistlike narrowing at the lower end of the esophagus above the level of the crura was found in all. It dilated under light pressure, and gave the impression of a light fibrosis. The only

other explanation which I have at present for it is that it is the permanent impression of the inflated lung tips. During the life of the individual such an impression occurs eighteen times a minute, accompanied by a momentary closing of the esophagus. Some people find it hard to attach as much importance to the action of the lung tips as I do. Constant watching of their action through the fluoroscope and seeing it constantly recorded on the X-ray film have convinced me that their action must play a part in the pathology of the lower end of the esophagus. (See Figs. 45-51.)

As the barium picture is practically the same at the subsequent visits of the patient, although the barium can be seen to go into the stomach in a larger stream, and the patient reports much less difficulty in swallowing, some of the old condition which existed before the dilatation has remained or returned. The waistlike constriction is too large to obstruct of itself, so I have come to the conclusion that the return of the closure is due either to spasm at the lower end of the esophagus or to a backward twist, preferring for myself the explanation of twist. The reader, of course, can take his choice. I will only remind him that such a backward bend can be present at birth and the stage all set for trouble.

The Operative Treatment of Cardiospasm.—Mild cases of cardiospasm can be managed by dilatation, preferably dilatation by sight. Where this is unsuccessful or the case has advanced to the stage marked by bend or twist, the condition must be dealt with by some form of surgery carried out through the upper abdomen. Incision into the musculature of the lower end of the esophagus, after the fashion of the treatment of pyloric obstruction at birth, has been tried successfully in this country. (Meyer.) In England, Grey Turner has twice made a lateral anastomosis between the mobilized lower end of the esophagus and the fundus of the stomach. In the next five or ten years I feel that some such operation will be the treatment for the long standing cases, especially those with twist.

I gave the conclusions of the paper at the beginning. In closing I shall not repeat them in detail. But I should like to remind you that the terminal portion of the esophagus in the resting position is closed. The deep vertical pleats and the abundant elastic tissue which is present at this point are the main

factors in bringing this about. The twist of the esophagus aids, so does outside pressure, especially the pressure of the lung tips. A deformity almost typical of an advanced case of cardiospasm can be present at birth. My case of cardiospasm showed a loss of the motor cells of Auerbach's plexus at the terminal portion of the esophagus, which is in line with the original observation of Stokes. Insufficient nerve supply would, of course, give altered and diminished muscular action. This must play a conspicuous part in cardiospasm, especially in the terminal stages. The esophagus is subject to degenerative diseases like arteriosclerosis. In one example of this condition the ganglion cells were found to be diminished. Important as the decrease of the ganglion cells is, and the altered muscular action resulting from this, a mechanical factor, the backward bend of the esophagus, to my mind, is equally important. It literally traps the lower end of the esophagus.

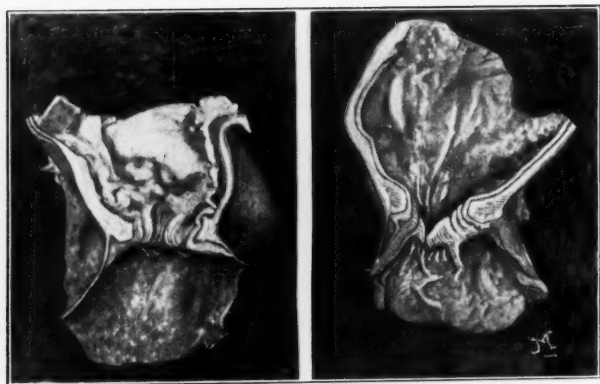


Fig. 1. Human; adult. Two specimens showing the pyloric sphincter.



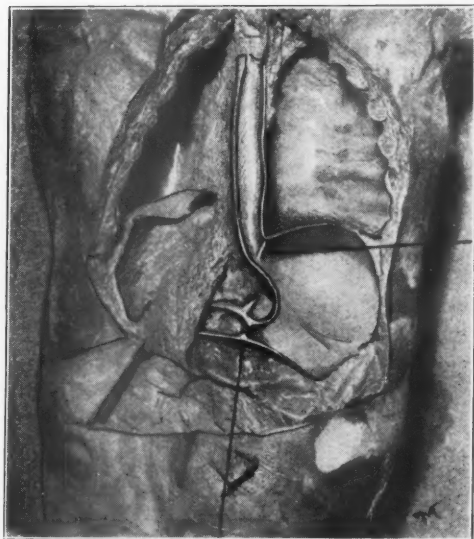
Fig. 2. Retouched photomicrograph showing the pyloric sphincter.
Human; adult.





Pyloric sphincter

Fig. 3. Baby dead at birth. The esophagus and stomach have been injected with wax. Both are normal. Notice the pyloric sphincter. See Fig. 4.



Pyloric sphincter

Fig. 4. Baby dead at birth. This is the same specimen shown in Fig. 3. The anterior wall of the esophagus and stomach has been removed. There is no thickening of the esophageal walls at the cardia to indicate a sphincter. There is an oblique fold on the posterior wall of the esophagus corresponding to the position of the left crus. Above this the esophagus tends to make a pocket. For many years the writer has noticed in specimens both the fold over the crus and the pocket above it. Notice the pyloric sphincter.



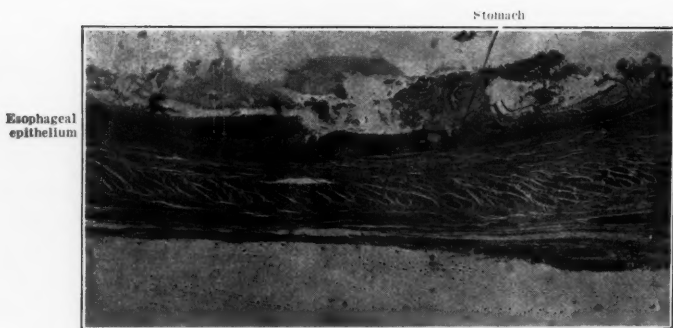


Fig. 5. Section of the lower end of the esophagus of a baby dead at birth. There is no anatomic sphincter.

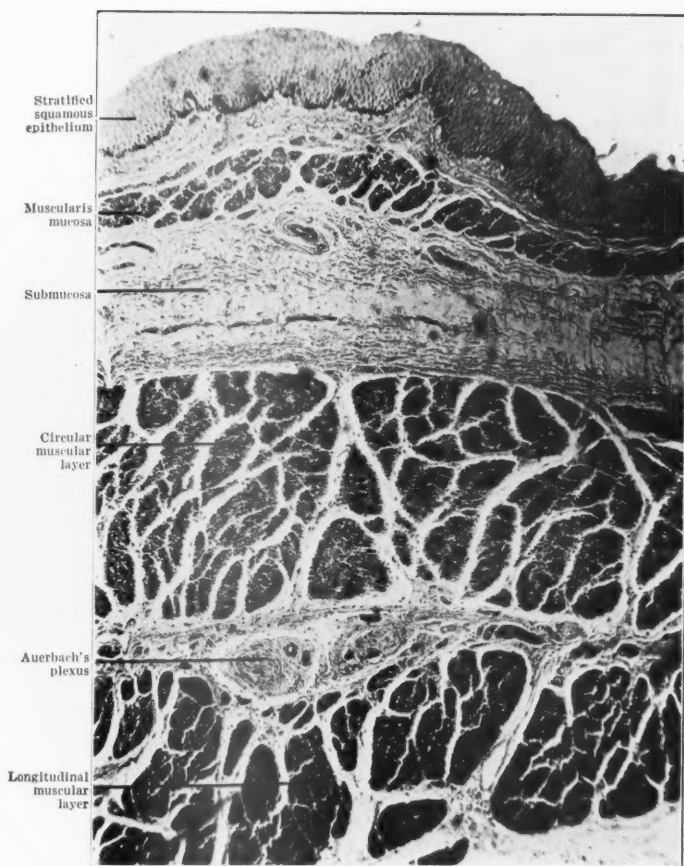


Fig. 6. Infant two years old. Section from the middle of the esophagus to show normal histology.



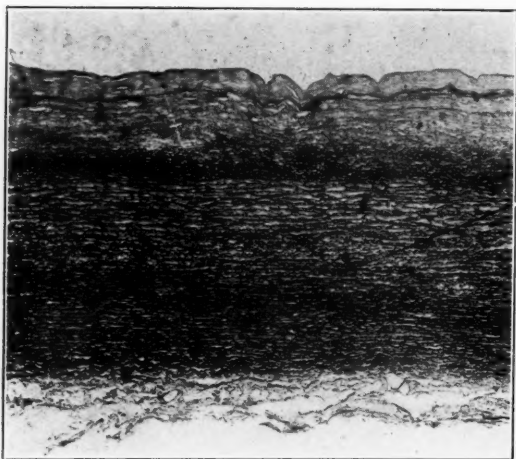


Fig. 7. Section of the descending aorta (human) stained to show elastic tissue.

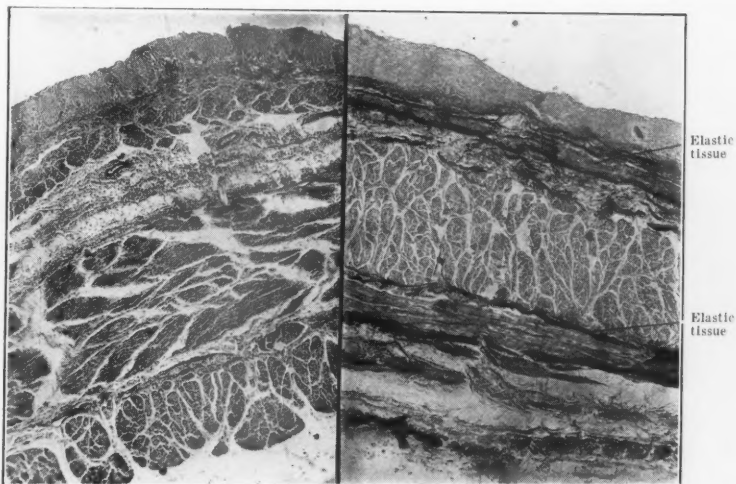


Fig. 8. Infant, two years. The first section is from the upper end of the esophagus, the second from the lower. In the first there is but little elastic tissue, in the second it is conspicuous in amount. Elastic tissue stain.



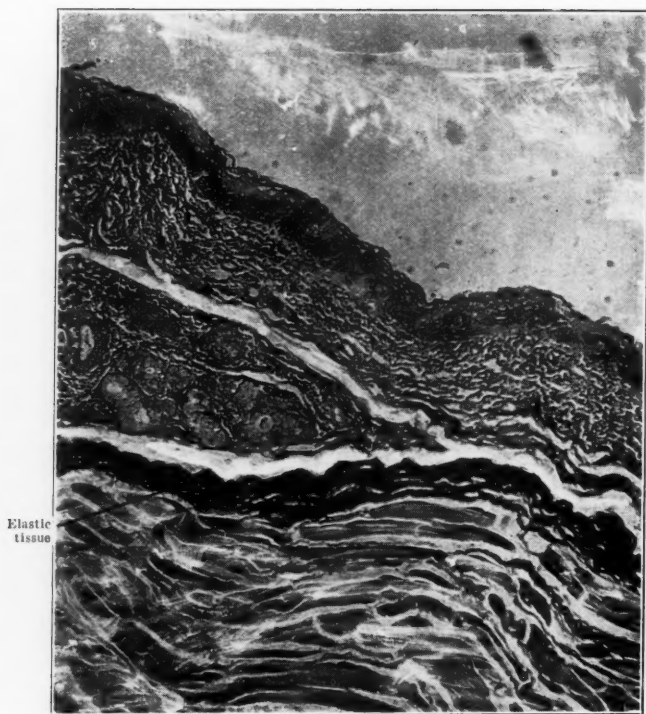


Fig. 9. Male, 61. Generalized arteriosclerosis. The lower end of the esophagus. Photomicrograph. The section shows a marked collection of elastic tissue between the submucosa and the circular layer of muscular fibres. Elastic tissue stain.

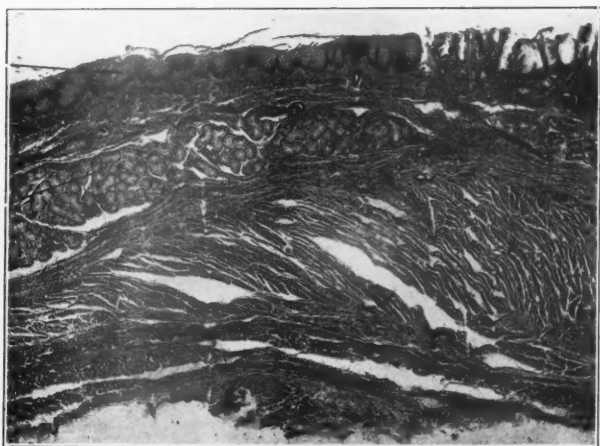


Fig. 10. Section of the lower end of the esophagus of a dog. Notice the large glands.

Epithelium
of
Esophagus



Mucosa
of
stomach

Fig. 11. Section of the lower end of the esophagus of a cat. Notice how abruptly the epithelium of the esophagus ends.



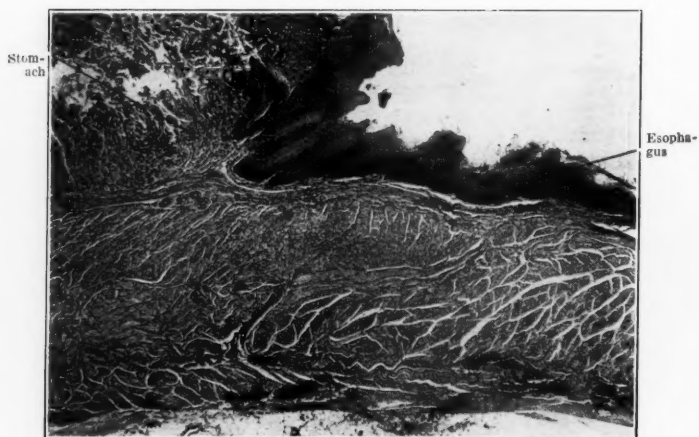


Fig. 12. Section of the lower end of the esophagus of a rabbit. The rabbit is said to have a cardiac sphincter. The writer's sections do not show one.

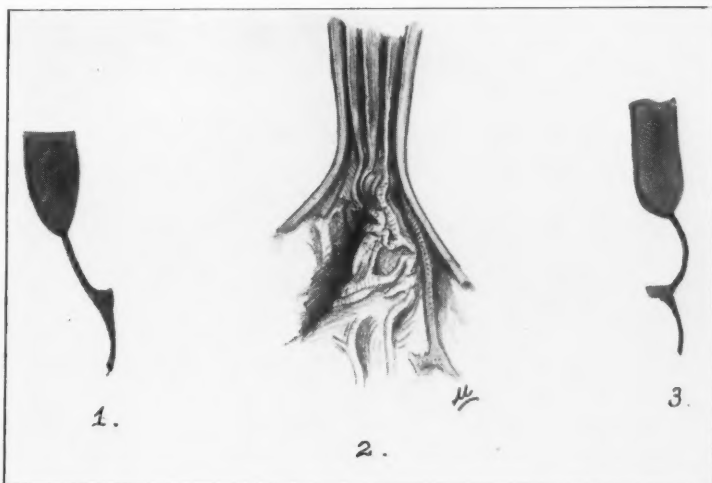


Fig. 13. The esophagus of a child. The illustration is to show the vertical pleats at the lower end of the esophagus. They are continuous for a short distance with the rugae of the stomach. On the anterior wall of the esophagus one or two pleats are continuous with long rugae which skirt the lesser curvature of the stomach. One of these rugae is shown in the illustration. In the resting or collapsed state of the terminal portion of the esophagus the barium runs in the grooves made by the pleats. This accounts for the pictures given by the barium meal. Figures 1 and 2 are such pictures. In these the esophagus is seen to come to a point. This is the point of occlusion due to the inflated lung tips. Below this there is a narrow ribbon of barium. This represents a channel between the pleats in the closed esophagus.

Where the esophagus ends and the stomach mucosa begins there is a slight ridge. Examination shows that this is due to a mounding of the mucous membrane of the esophagus. Although to the naked eye the esophageal mucous membrane seems to stop at this ridge microscopical examinations shows that it may extend fully 5 cm. below this point.



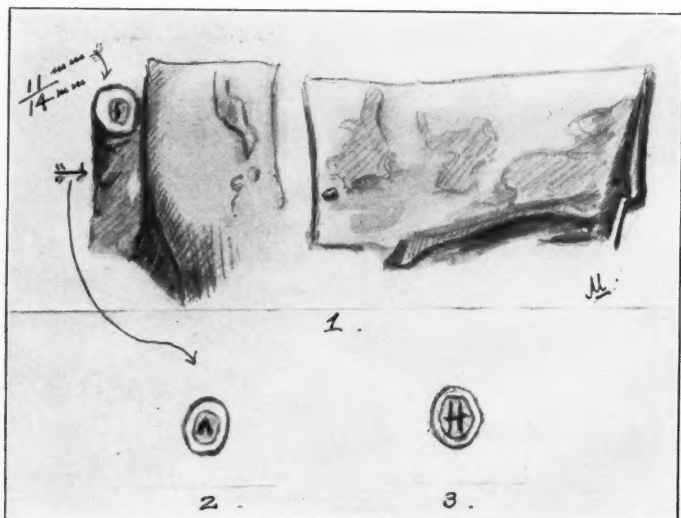


Fig. 14. Male, 61. Generalized arteriosclerosis. Specimen showing the lower portion of the esophagus and a section of the descending aorta. The second figure of the upper row is the aorta laid open to show patches of arteriosclerosis.

The esophagus in the first figure is seen to be a narrow closed tube. The closure is due to the vertical pleats. Figures 2 and 3 show the character of the pleating at lower levels. In Figure 3 that there are two deep vertical pleats, one on the anterior wall of the esophagus, the other on the posterior wall. Notice also how these two pleats practically close the esophagus. The writer believes that this is the normal resting condition of the terminal or pleated portion of the esophagus.

See Fig. 13.

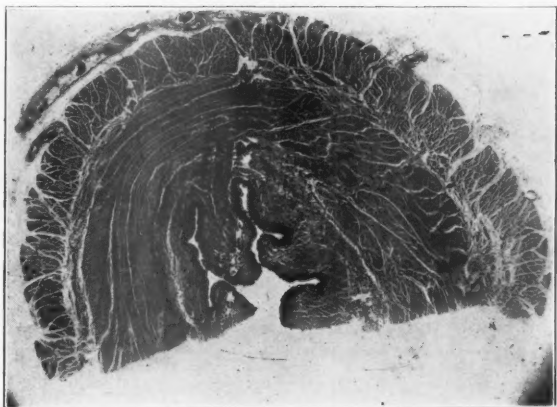


Fig. 15. Cross section of one-half of the lower end of the human adult esophagus showing the normal pleating.



Fig. 16. Male, 25. Cardiospasm one year. X-ray film of the lower end of the esophagus.

The dilated esophagus comes to the typical point. Below this there is a narrow spindle shaped ribbon—the pleated portion of the esophagus—which merges into the barium shadow along the lesser curvature of the stomach. The pleated portion in many films appears as a ribbon of uniform diameter. At times the film shows peristalsis acting at this point.

This picture with slight modifications in the shape of the pleated portion may be taken as the characteristic one obtained in cases of cardiospasm of moderate severity.

See Fig. 13.

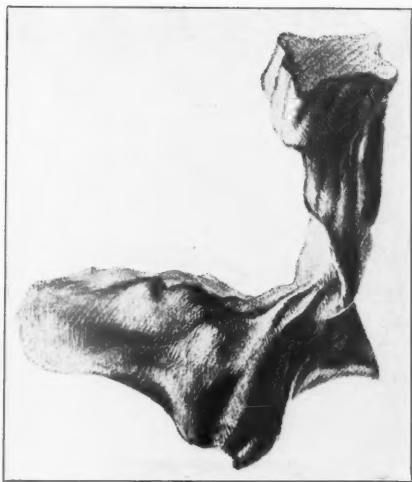


Fig. 17. Drawing of a reconstruction of the lower end of the esophagus from frozen sections. Adult.

The drawing shows that the terminal portion of the esophagus consists of a vertical and a horizontal portion. The vertical part comes to a point and its axis changes from transverse to anteroposterior. The horizontal portion or arm is very much pleated. This part the writer believes to be closed in the resting condition. The closure is due mostly to the pleats. See Figs. 13-14-16.

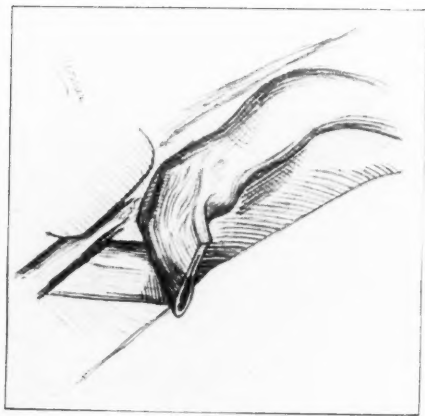
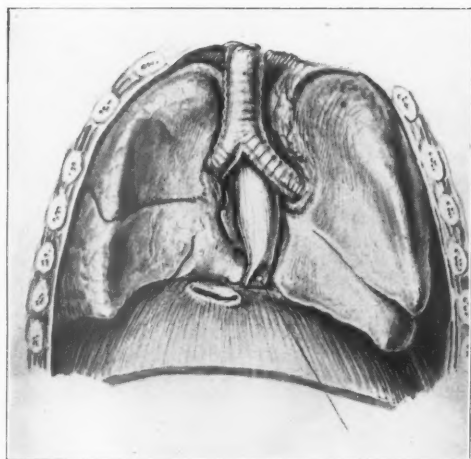


Fig. 18. Drawing of the terminal portion of the esophagus of an adult cadaver. The esophagus was not injected. The specimen shows the twist of the terminal portion of the esophagus. Notice how the left crus lies behind the horizontal arm or pleated portion of the esophagus. The anterior crus has been mostly cut away. Above the edge of the left crus there is a globular dilatation of the esophagus.



Impression of left lung tip.

Fig. 19. Drawing from a cast of the trachea, lungs and esophagus of a baby dead at birth.

The drawing shows the relation of the lung tips to the terminal portion of the esophagus. The impression of the left lung tip on the esophagus stands out clearly.

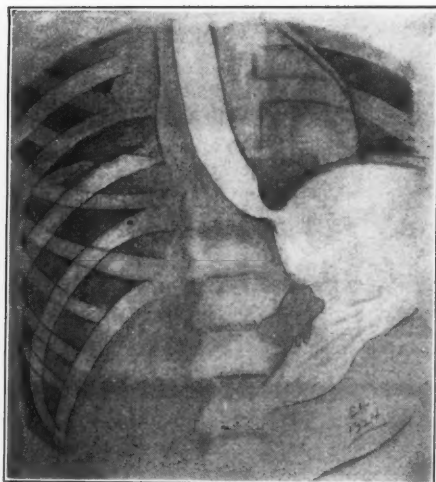


Fig. 20. Retouched tracing from X-ray film. Adult. The film was taken in full inspiration.

The film shows a very marked impression of the left lung tip on the left side of the terminal portion of the esophagus.

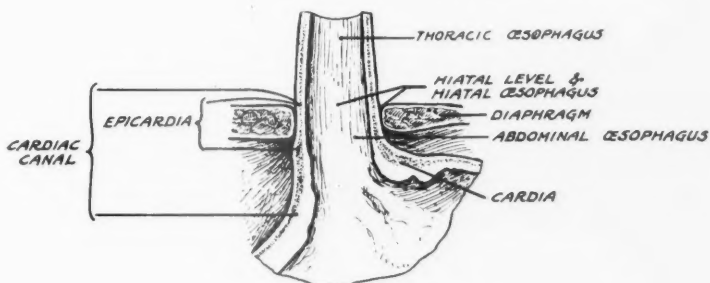


Fig. 21. Retouched tracing from X-ray film. The diaphragm is down and the inflated lung tips have momentarily closed the esophagus. The narrow strip extending below the closed and distended esophagus represent barium caught in the vertical folds of the esophagus.

See Figs. 13-14.



Fig. 22. Female, 40 years. Cardiospasm one year.
The barium bougie is in place. The diaphragm is down. There is a waist like constriction of the terminal portion of the esophagus above the dome of the left half of the diaphragm. The most marked portion of the constriction is on the left and corresponds to the shape of the left lung tip.



—Cardiac Canal with Names.

Fig. 23. Dissection by Professor Shattock to show the normal cardiac canal and orifice. Kelly.

Mich

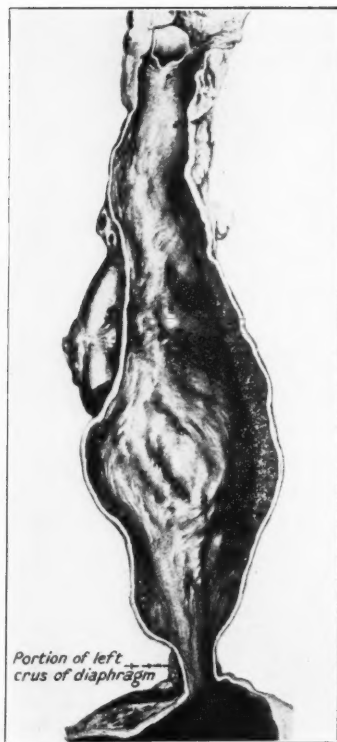


Fig. 24. Female, 56 years. Dysphagia 20 years.
 Autopsy specimen of the esophagus from a patient who suffered from
 cardiospasm. Illustration from Kelly.
 Notice the dilated upper portion of the esophagus, and the narrow,
 tubular terminal portion.

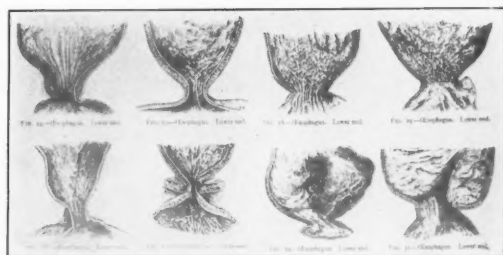


Fig. 25. Drawings of the lower end of the esophagus from autopsy specimens in eight cases of cardiospasm. The cases were reported by Moore. The figures are from Moore and are here taken from an article by Kelley who quoted them from Moore.

The first four drawings—to the middle of the illustration—show the lower end of the esophagus narrow and tubular. This tubular narrowing Moore calls the cardiac canal.

In the last four drawings there is no cardiac canal, only a fibrous ring. The stenosis of the esophagus is not as marked as in the first four cases.

The cases of cardiospasm which die of starvation are found, according to Moore, in the first group of four.

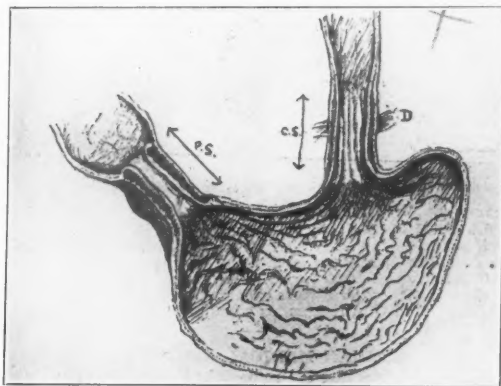


Fig. 26. Diagram from Hurst to show the cardiac and pyloric sphincters.



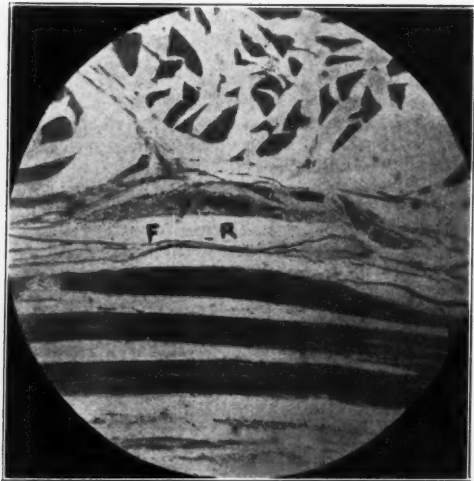


Fig. 27. Drawing from Hurst. Section of the esophagus from a case of cardiospasm.
The drawing shows infiltration and fibrosis of Auerbach's plexus.

Bend at
terminal
portion



Fig. 28. Retouched tracing from X-ray film. Mac D., 55; male. Cardiospasm 15 years. Death from tuberculosis of the lungs. The film shows the typical picture of an advanced case of cardiospasm. A No. 35 French elastic bougie would pass easily. See Figs. 29-30-31.

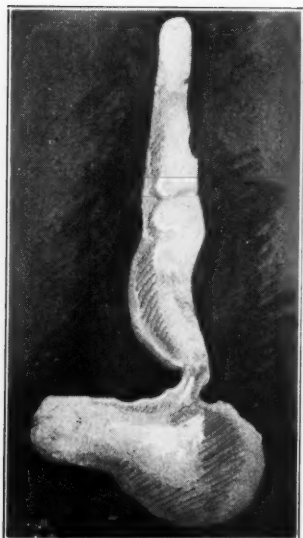


Fig. 29. Mac D., male; 55. Cardiospasm 15 years.

Cast of esophagus and stomach.

The esophagus is much dilated except at the lower end where there is a narrow tubular portion about an inch long. There is a marked backward bend of the esophagus above the tubular portion.



Fig. 30. Mac D., male; 55. Cardiospasm 15 years. Cast of esophagus and stomach.

The terminal portion of the esophagus seen from the right and from the left. Notice the backward bend.

The writer feels that this backward bend of the terminal portion of the esophagus plays an important part in producing the obstruction present in cases of cardiospasm. See Figs. 28-29.





Fig. 31. Mac D., male; 55 years. Cardiospasm 15 years. Death from tuberculosis of the lungs.
Photograph of esophagus and stomach. Notice the dilated upper portion of the esophagus and the narrow terminal portion.
See Figs. 28-29-30.

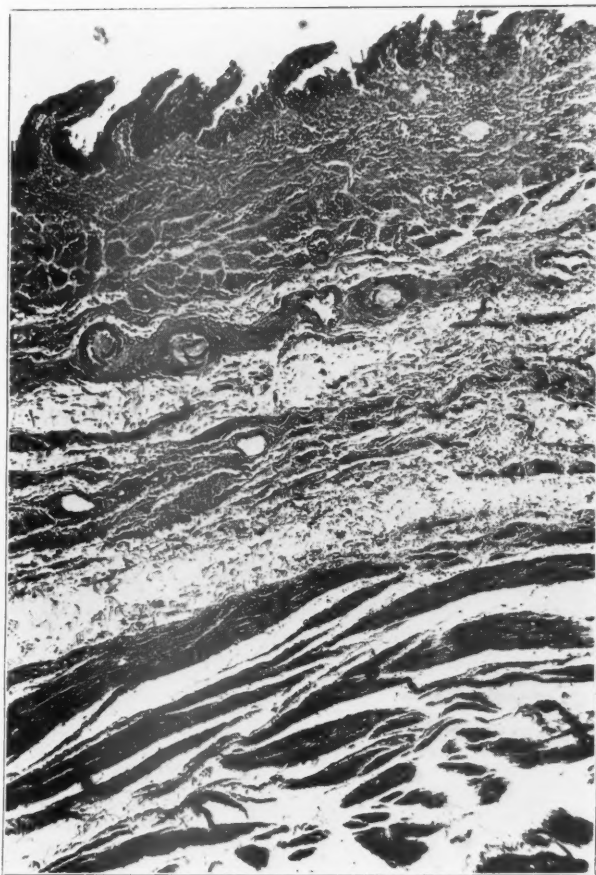


Fig. 32. Mac D., male; 55 years. Cardiospasm 15 years. Death from tuberculosis of the lungs.

Section of the esophagus showing superficial infiltration with lymphocytes and plasma cells. There is an increase of connective tissue in the infiltrated area.

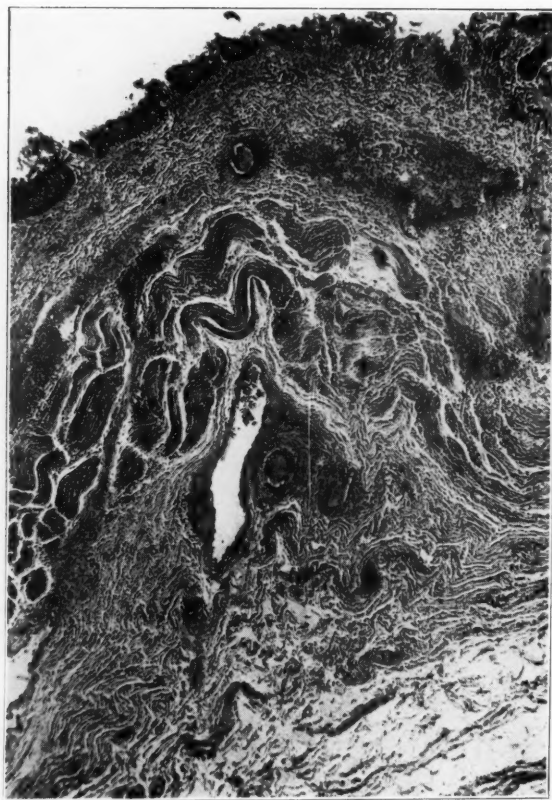


Fig. 33. Mac D., male; 55 years. Cardiospasm 15 years. Death from tuberculosis of the lungs.

Section of the esophagus showing superficial infiltration with lymphocytes and plasma cells. The infiltration has penetrated the muscularis mucosa and appears beneath it round the blood vessels.

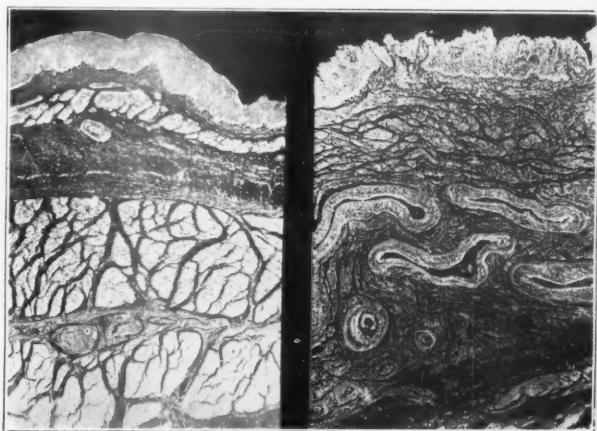


Fig. 34. The first illustration is a section of the esophagus of a child of two years. It shows but one blood vessel in the submucosa. The second illustration is a section of the esophagus from the writer's case of cardiospasm (Mac D., 55 years). Notice the increased number of blood vessels, their great size, and thick walls. Practically the same increase in the number of the blood vessels, and the same enlargement, was found in the sections of the esophagus of a man who showed at autopsy extensive generalized arteriosclerosis.



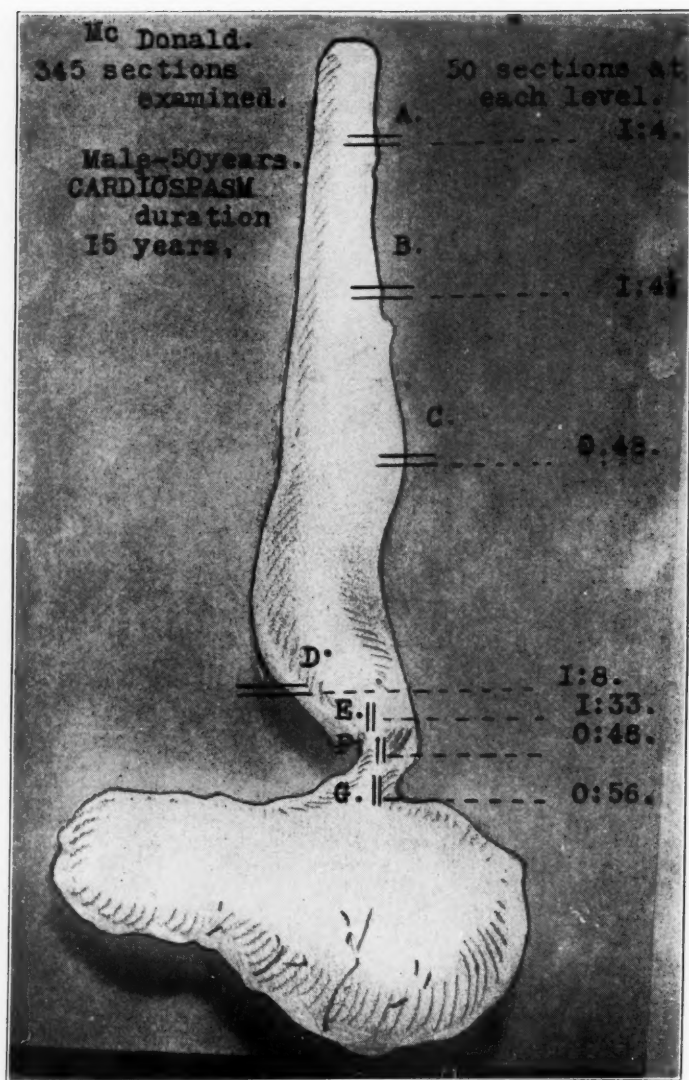


Fig. 35. Tabulation of the ganglion cell count in Auerbach's plexus at seven different levels in the writer's specimen from a case of cardiospasm. At the terminal portion of the esophagus there were no ganglion cells in one hundred sections. In an infant the average number of ganglion cells to a plexus is 27.

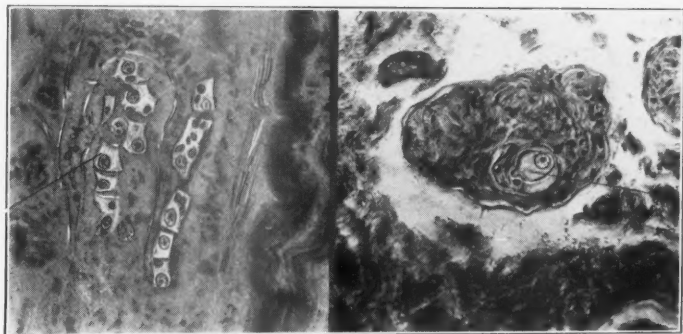


Fig. 36. Photomicrograph.

The first illustration is from the esophagus of an infant two years old. It shows one ganglion with twelve ganglion cells.

The second illustration shows an Auerbach's plexus from the writer's case of cardiospasm. But one ganglion cell remains. The reader is reminded that in this specimen at the lower end of the esophagus no ganglion cells were found in one hundred sections.



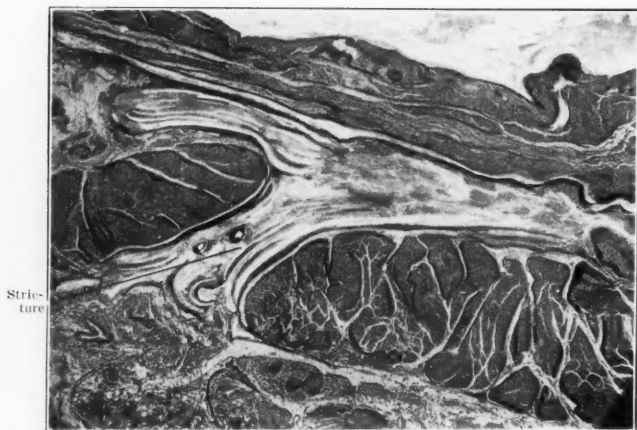


Fig. 37. Human adult, male; 50 years.

The patient had a stricture of unknown etiology at the lower end of the esophagus for ten years. He was dilated successfully under ether. Three years later a second dilatation was necessary. During this the esophagus was perforated by the bougie and he died of septic pleurisy and peritonitis.

The microscopical section shows the stricture. It is confined mostly to the region occupied by the circular layer of muscular fibres. The esophagus has lost its epithelium and the mucosa and submucosa are filled with connective tissue.

This stricture case is in marked contrast to the case of cardiospasm in which the infiltration and the increase of connective tissue was confined chiefly to the mucosa and submucosa.

See Fig. 32.

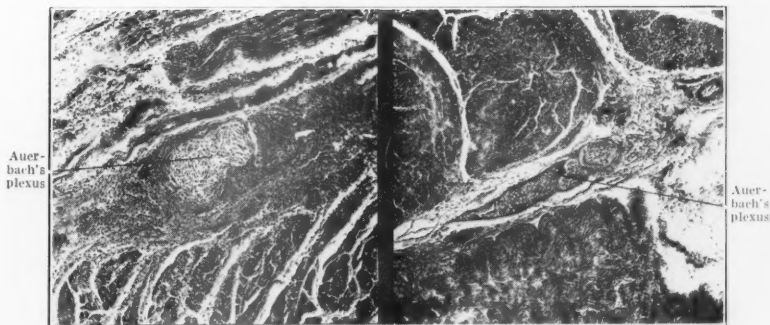


Fig. 38. Human, adult; 50 years. Photomicrograph showing Auerbach's plexus.

The patient died of septic pleurisy and peritonitis after perforation of the esophagus by a bougie.

The first illustration shows a plexus surrounded by a shower of leucocytes and plasma cells but not as yet invaded by them. In the second illustration two-thirds of the plexus has been invaded.

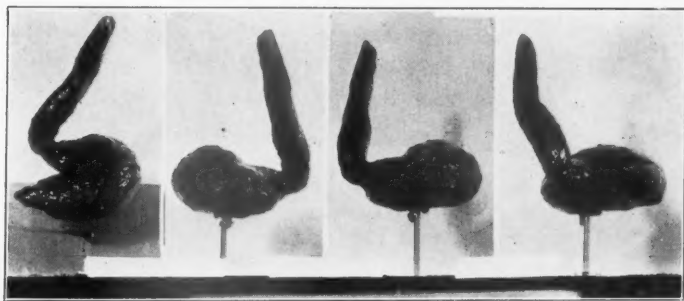


Fig. 39. Four views of a wax cast of the esophagus and stomach of a baby dead at birth. The stomach has swung upward until its long axis is horizontal. The esophagus is dilated, has an S-shape curve to the right, and the lower end has a pronounced backward bend.

See Fig. 41.

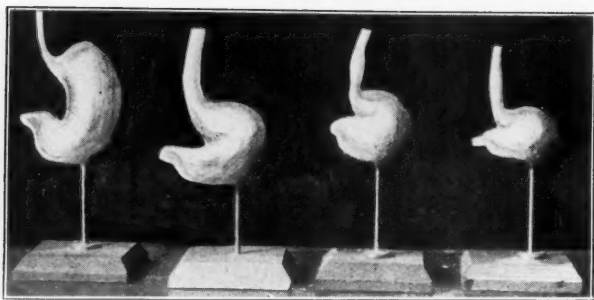


Fig. 40. Four babies dead at birth.

The esophagus and stomach before removal were injected with wax.

The illustrations show the gradual upward swing of the stomach until its long axis becomes in the last specimen horizontal. This position of the stomach tends to produce a kink at the lower end of the esophagus.

The usual conception is that a baby's stomach is vertical. The upward swing shown in the illustrations is not, however uncommon.



Atken

Fig. 41. Baby dead at birth.

Esophagus and stomach injected with wax. The upper part of the esophagus is flattened from before backward by the pressure of the trachea. Only the lower half is cylindrical.

The terminal portion of the esophagus bends on itself at an angle of 90° to reach the stomach.

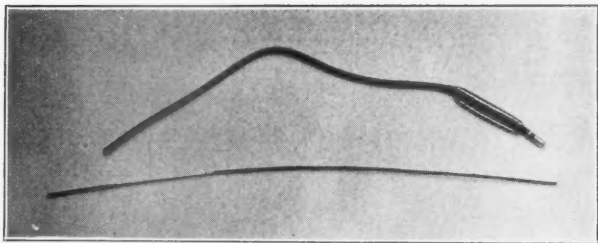


Fig. 42. The writer's diagnostic barium bougie.

The upper figure is the bougie, the lower one the whalebone staff for introducing it. The bougie consists of a red rubber tube the size of the usual stomach tube. Fastened to the end of the tube is a blunt metal tip half an inch long and a quarter of an inch in diameter. The upper end of the metal tip has a hole bored in it to take the end of the whalebone staff and to keep it in place during the introduction of the bougie. Near the top of the metal tip there is a groove to take the thread which ties on the lower end of the rubber bag. Inside the rubber tubing at the point where the shank of the top of the rubber bag is to come there is a thin metal ring with a groove round the middle of it. This is to take the thread which ties the upper end of the rubber bag.

In the lower end of the rubber tubing at a point about under the center of the rubber bag four or five holes are punched. These allow the air which is forced into the tubing to inflate the bag. A thin rubber bag tied over the lower end of the tubing completes the main part of the apparatus. The bag is moderately inflated with air and kept distended while four barium lines are painted on it, using equal parts of barium powder and ordinary rubber cement. The lines should divide the circumference of the bag into four equal parts. They dry in twenty minutes to half an hour and will stay on the bag some six weeks. Two of the lines show in the illustration. In order to keep the lines from being washed or scrubbed off during the use of the bougie a second rubber bag is tied over the first one.

To a metal Y-tube the air bulb from a blood pressure apparatus and an air pressure gauge registering up to fifteen pounds and with a radiant dial are attached ready for coupling up with the bougie after it has been passed.

The bougie is greased and the staff also. The patient's pharynx is sprayed with a half of one per cent solution of cocaine. The patient is placed before the fluoroscope and the fluid level in the esophagus obtained by giving her—most of my patients have been women—a part of a glass of barium. A quick examination of the chest, the lower end of the esophagus, the action of the lung tips and the movement of the diaphragm is made, and then the barium which has accumulated in the esophagus is hurried on into the stomach by giving the patient a fizzing Selditz powder to drink. The writer hit upon this expedient for emptying the esophagus some two years ago and has since found it very much worth while. Besides emptying the esophagus and so making the passage and the retention of the bougie less messy, it gives a large gas bubble in the stomach against which the barium picture of the lower end of the esophagus stands out clearly.

The preliminary fluoroscopic examination over, the patient is seated in a chair and the diagnostic bougie is passed by touch. It usually halts a little at the lower end of the esophagus and then with a slight jump forward as if it passed an obstruction enters the stomach. If the bougie does not pass with the use of almost no force and a little manipulation the patient is placed before the fluoroscope and an attempt is made to manipulate the bougie into the stomach by sight. If the bougie does not then pass readily the attempt is given up. Usually, however, it does pass and when the patient is placed before the fluoroscope the bougie is found well down in the stomach. This being the case, the bougie is drawn up into place by sight, half of the bag being in the esophagus and half in the stomach. At this point the air pressure apparatus is connected to the bougie and the bag is inflated. So far the writer has used from four to six pounds of pressure. The thin rubber bag can be made to dilate in the stomach like a balloon. This is in striking contrast to the smaller esophagus above. While the patient is still standing before the fluoroscope four films are taken, two in the anteroposterior and two in the right lateral position. In each position one film is taken in full inspiration, and one in full expiration.



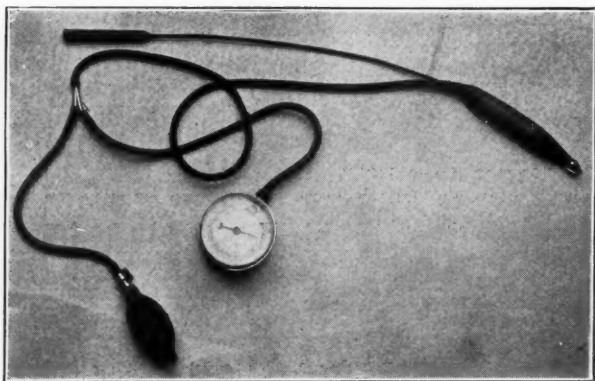


Fig. 43. In order to be able to use more pressure for dilatation than the diagnostic bougie will give, the writer uses the Sippy bag. The illustration shows the bag with the air pressure apparatus attached.

Four barium lines are painted on the bag and the silk cover drawn on. In order to take away the harshness of the corners of the silk cover a rubber bag is tied in place over it. The silk cover keeps the Sippy bag from dilating unduly. This apparatus is capable of exerting a pressure of thirteen to fifteen pounds.

The Sippy bag can be used for diagnostic purposes as well as for dilating. The thin bag of the diagnostic bougie, however, as it balloons so readily in the stomach, gives a more dramatic picture.

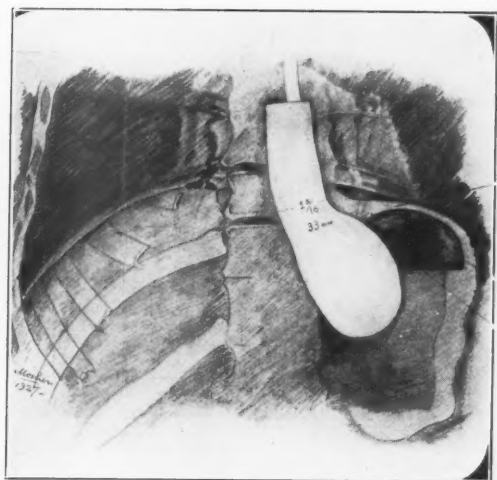


Fig. 44. Adult, male; 30 years. Normal esophagus. Retouched tracing from X-ray film. The diagnostic barium bougie is shown in place. It gives the normal transverse diameter of the terminal portion of the esophagus to be 33 mm. The anteroposterior diameter is practically the same.

In this case when the diaphragm was down there was but a slight narrowing of the esophagus.



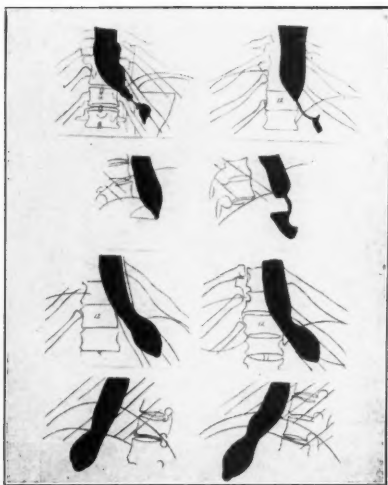


Fig. 45. Male, 30 years. Cardiospasm one year. The upper four figures are the barium meal findings, the lower four the findings with the barium bougie. Figs. 1-2, 4-5 are films taken in the anteroposterior position. Figs. 3-4, 7-8 are from films taken in the lateral position. Figs. 2-4-6-7 were taken with the diaphragm down.

The films taken with the barium meal give the impression that the esophagus is tightly strictured. The lower four figures from films of the barium bougie in place show that this is not the case.

Notice the impression of the left lung tip shown in Figs. 6-7-8.

Notice also how the terminal portion of the esophagus—the pleated portion—is practically closed.

See Figs. 2-4 of this plate.



Fig. 46. Female, 40 years. Cardiospasm one year.
Lateral film. The diagnostic barium bougie is in place. It shows a long waist like constriction of the terminal portion of the esophagus.



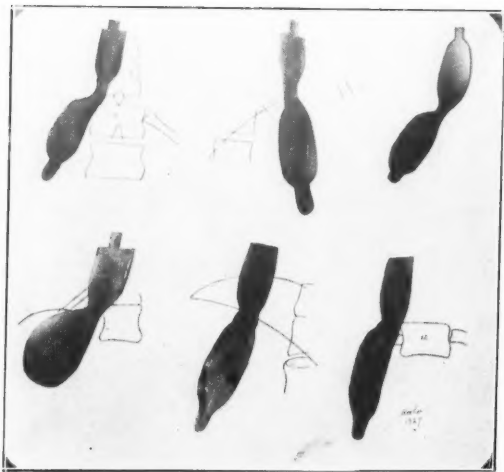


Fig. 47. Retouched tracings from X-ray films of four cases of cardiospasm. Figs. 1-2 are from the same patient. So are Figs. 5-6.

The films show the variations in the size of the waist like constrictions shown by the barium bougie. In these cases the esophagus dilated to normal size with a pressure not exceeding four pounds in three. The one which did not is No. 4 in the series.

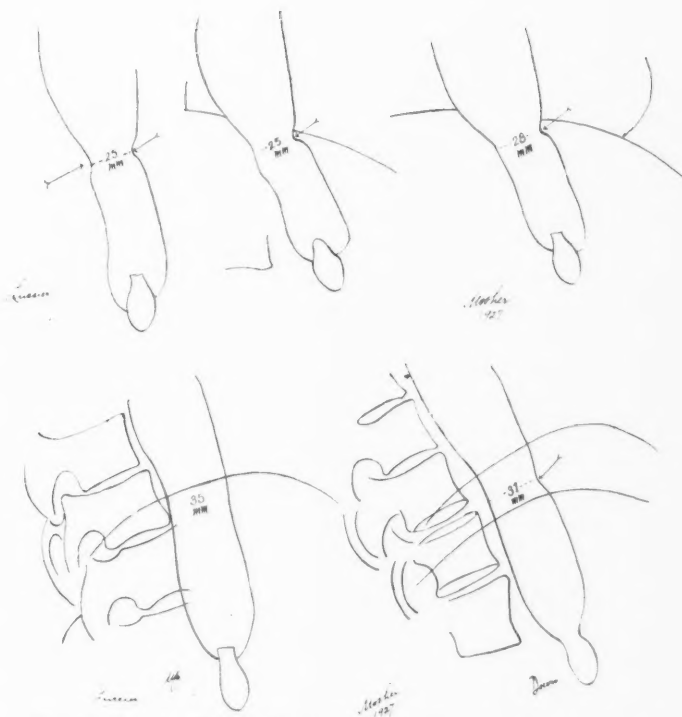


Fig. 48. Female, 35. Cardiospasm nine years.

Tracings from X-ray films showing the findings of the barium bougie. The first three films were taken in the anteroposterior position and the last two in the right lateral.

Notice how in the fourth figure the esophagus has dilated to the normal diameter (33mm).

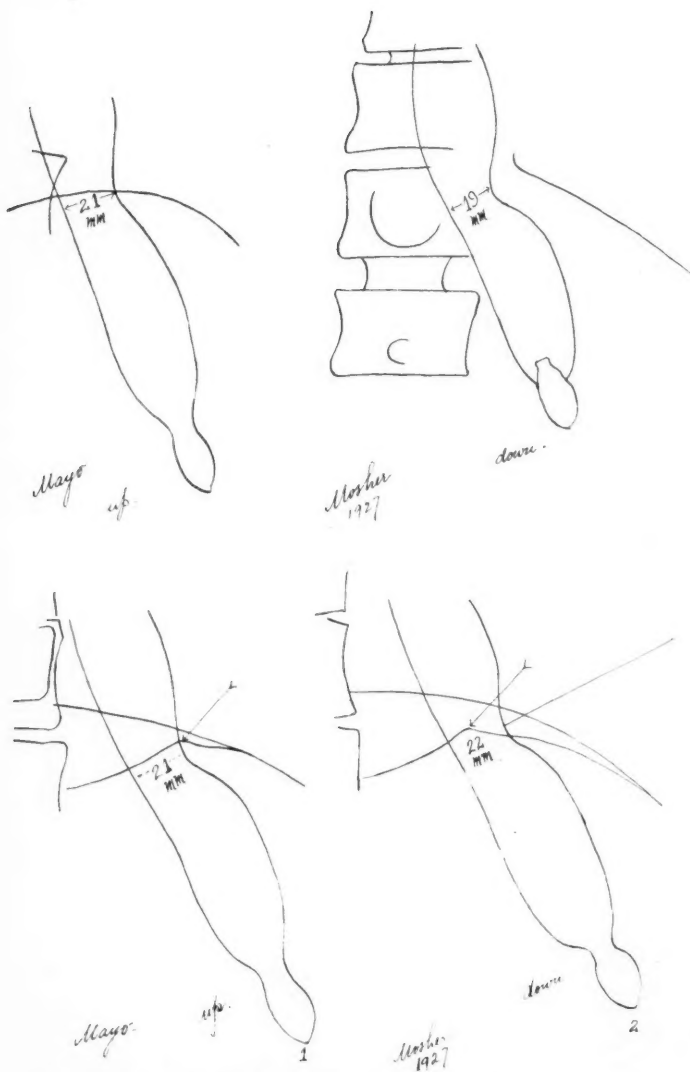


Fig. 49. Male, 32. Cardiospasm four years.
 Tracings from X-ray films showing the findings of the barium bougie. The first two films were taken in the anteroposterior position, the second two in the lateral position. Notice in the second figure the marked impression of the left lung tip. In the third and fourth figures the diaphragm shows a pronounced adhesion indicating previous pathology in the chest.
 This case had a gastrostomy and retrograde bouginage. It is one of the two cases which have not dilated to normal.

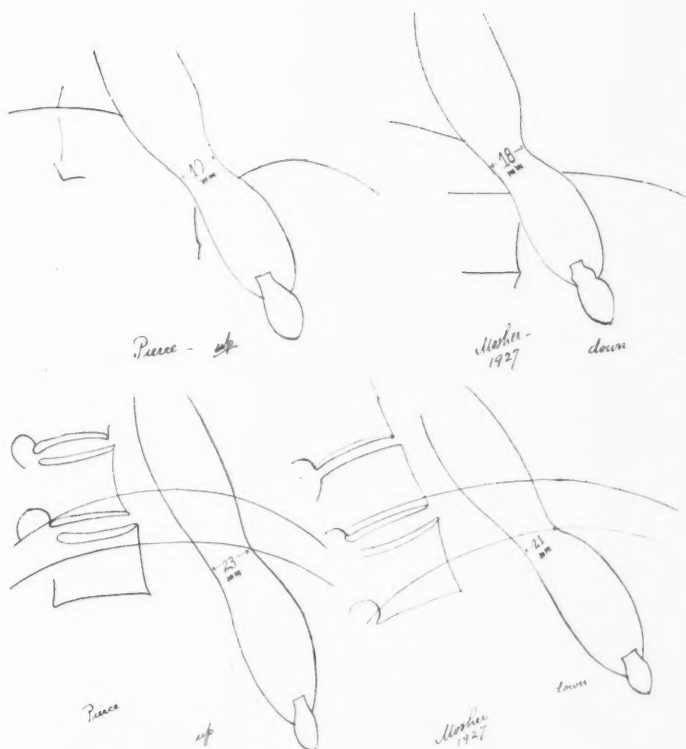


Fig. 50. Female, 45 years. Cardiospasm two years.

Tracings from X-ray films showing the findings of the diagnostic bougie. The first two figures are from films taken in the anteroposterior position and the second two from films taken in the lateral position.

This patient has been dilated twice and the esophagus so far has not dilated to normal. This case is one of two in a series of nine which has not dilated to normal.

Notice in the second figure the marked impression of the left lung tip. See Fig. 49.

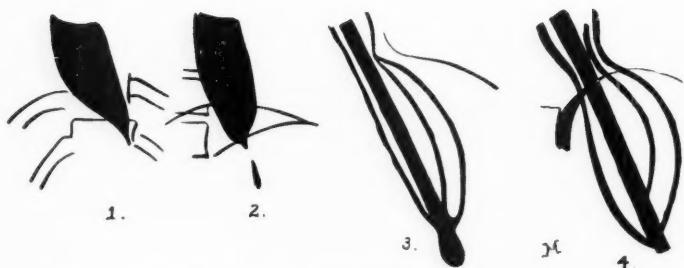


Fig. 51. Female, 20 years. Cardiospasm one year.

Figs. 1-2 are the barium milk findings.

Figs. 3-4 are the diagnostic bougie findings.

Figs. 1-3 are anteroposterior films; 2-4 lateral films.

Notice in 3 the very marked notch on the left wall of the esophagus. This is opposite the left lung tip.

In 4 the narrowing is seen to extend—waist like—all round the esophagus. It is more marked in front than behind.

These contrasted illustrations show the added information furnished by the diagnostic bougie over the barium meal.

IV.

PEDIATRIC ASPECTS OF OTOLARYNGOLOGY.*

By McKIM MARRIOTT, M. D.,

ST. LOUIS.

One of the most important trends in pediatrics during recent years has been a growing appreciation of the importance of infections of the ear, nose and throat in the causation of general diseases and nutritional disturbances. This has been the result of a larger measure of cooperation between otolaryngologists and the internists. The time is passing when the pediatrician merely glances at the throat as part of the general examination of a child, sees enlarged tonsils, obtains a history of mouth breathing and sends the child to the otolaryngologist for a tonsillectomy and adenoidectomy. The otolaryngologist is no longer content to remove tonsils and adenoids in a routine manner without knowing something about the patient's general condition and the indications for surgical procedure. In the better clinics it is now the custom for the pediatrician and the otolaryngologist to examine the patient together to discuss all of the features which may have influenced the child's condition and in this way to arrive at a proper appraisal of the significance of pathologic conditions in the ears, nose or throat.

It is the purpose of the present paper to point out some of the ways in which such cooperative effort has resulted in better understanding of diseases in infancy and childhood.

One of the difficult problems of the pediatrician has been the artificial feeding of infants and the treatment of gastrointestinal and nutritional disorders. We have learned how to render food free from harmful bacteria which might set up gastrointestinal disturbances, yet, nevertheless, many infants, when fed in a manner theoretically ideal, still fail to thrive or develop vomiting and diarrhea, and a certain number of them succumb.

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A search for the causes of failure in these cases was for many years largely fruitless. Nothing significant was revealed at the autopsy table, there were no changes found in the gastrointestinal tract or in any of the organs of the body. The bacteriology of the intestinal tract, except in cases of bacillary dysentery, did not differ from that of normal infants. A search for poisonous products of the metabolism of food was unsuccessful. Chemical examination of the blood revealed only such changes as could be considered secondary to starvation, vomiting and water loss. In these cases the key to the solution of the problem lay in the clinical observation that the majority of infants who failed to do well despite proper feeding suffered from infections of more or less severity in the nose, throat or ears. Some pharyngitis was commonly observed. The ear drums were occasionally reddened and paracentesis at times revealed serum or pus.

For many years there was a tendency to regard these infections entirely as secondary manifestations of a poor nutritional condition. Upon more careful observation, however, and the obtaining of more detailed histories, it became evident that these infections often preceded nutritional or gastrointestinal disturbances. In a certain number of infants there was observed a gradual decline in nutrition, as the result of inadequate food in the absence of any evidence of infection. Such infants, because of their poor nutritional condition, seemed especially susceptible to infections. With the onset of a slight rhinopharyngeal infection there was often observed a rapid decline in weight, and at times diarrhea and vomiting. These infants, already in poor nutritional condition, showed a more severe reaction to infection than infants previously well nourished. In those cases in which it was possible to clear up the infection by local treatment of the rhinopharynx and by myringotomy, there occurred simultaneously an improvement in the infant's nutritional condition and a cessation of the gastrointestinal symptoms. Treatment of the infection in these cases exerted a far greater influence than any changes in the character of the food. It was, in fact, found unnecessary to make any changes in the feeding other than to give an adequate diet in those cases in which the food was quantitatively or qualitatively insufficient.

In a considerable proportion of the cases, however, the symptoms persisted despite the methods of treatment adopted. The pharyngitis did not clear up, and the ears, after paracentesis, continued to discharge and were the seat of repeated reinfection. In some of these cases removal of the adenoids and tonsils was followed by subsidence of the infection. Repeated blood transfusions served to improve the nutrition and apparently to increase resistance to the infections, but the results were not uniformly successful. In a good many infants the temperature persisted, severe gastrointestinal disturbances continued and there was rapid loss of weight and ultimately death. It seemed unlikely, in these cases, that a superficial pharyngitis or a freely draining middle ear could account for the high temperatures and severe symptoms. The autopsy findings were, in general, entirely negative, except in those cases in which a special examination of the ears, nose and throat were made, when it was found that practically all of these infants had definite evidences of infection in the mastoid antrum. Careful examination revealed the fact that the antrum was often completely blocked off from the remainder of the tympanic cavity by obstruction at the aditus.

The finding of pus in the mastoid antra of infants dying from nutritional disturbances was not a new observation. As far back as 1684, Du Verney¹ recognized at autopsy pus in the mastoid antra of young infants, but considered this to have originated postmortem. In 1921, Maurice Renaud² found pus in the mastoid antra of 70 infants dying of gastrointestinal and nutritional disorders. Byfield³ and Floyd,⁴ working in conjunction with Dean of Iowa City, noted the frequent occurrence of mastoid antrum infections in infants suffering from the symptoms mentioned. Our own experience dates from 1923, when we examined, at autopsy, the mastoids of a number of infants dying of severe nutritional disturbances, and found pus. These findings naturally led to the suspicion that the antrum infections were the cause of the continuation of the severe symptoms. Drainage of the infected antra during life was therefore carried out. The simple operation of antrotomy, or postauricular drainage, was, in many instances, followed by an extraordinary change in the general condition of the infants. The results were better in those cases in which the

infants were treated early in the course of the disease. In favorable cases the temperature fell, diarrhea and vomiting ceased without any change in the character of the feedings. It should be emphasized that in most instances there were no external evidences of mastoid involvement. There was no swelling, redness or tenderness over the mastoid. On otologic examination there was usually seen some sagging of the posterior superior portion of the drum and adjacent canal wall.

The decision to operate on these infants depends as much upon general medical as otologic indications. It is essential that other possible causes of gastrointestinal disturbances should definitely be ruled out. The details of the operation have been adequately described by Lyman and Alden⁵ and need not be referred to at this place.

Infections of the mastoid antrum may be due to a variety of organisms, and the symptoms observed are, to a certain extent, dependent upon the type of organism present. In our own series of cases, we found, during one year, the most frequently occurring organism to be a hemolytic streptococcus. A number of the strains isolated were studied in detail and were found to be strong toxin producers, the organism in this respect being indistinguishable from the streptococcus of scarlet fever. In some of our most recent cases we have found pure cultures of the colon bacillus, and further tests have shown the strains isolated to be very active producers of histamine. This latter finding is of interest because of the known effect of histamine, when injected, of causing the symptoms of vomiting, diarrhea and hydrolability. Other organisms which have been frequently found are the pneumococcus, B. of Morgan, and B. pyocyaneus. It has been our experience that staphylococcus infections of the mastoid give rise to quite a different variety of symptoms. The gastrointestinal symptoms are not marked, but there are likely to be local manifestations of mastoid involvement with subperiosteal abscesses. It is especially interesting to note that the organisms found in the mastoid antrum at the time of the operation and at autopsy have very frequently been quite different from those found in the middle ear, even when cultures of the latter were taken at the time of the first paracentesis.

Infections of the middle ear and mastoid giving rise to nutritional and gastrointestinal disturbances are much more frequent during certain years than others. Such infections seem to come in epidemics. Whether or not the infections persist and whether or not surgical interference is necessary depends a great deal upon the previous condition of the infant. A well nourished breast fed baby contracting a rhinopharyngeal infection or an otitis media is likely to recover in a short while, and to require very little treatment. A malnourished, underfed infant, on the other hand, may, with the same type of infection, be sick for days or weeks and finally have to be subjected to the operation of antrotomy.

Infections of the ear, nose and throat, causing gastrointestinal and nutritional disturbances, are probably no more common at the present time than they have been in past years. Their seeming preponderance is the result of more accurate diagnosis rather than of more frequent occurrence. Disturbances of infants, due to improper feeding, or to bacterially contaminated food, are certainly becoming less frequent, so that the proportion of cases in which the primary factor is infection is constantly rising. In our own experience over 85 per cent of all gastrointestinal and nutritional disturbances of infants in recent years have been due primarily to infections in the ear, nose and throat. In warmer climates and in regions where the milk supply is questionable and where infant welfare work is not well organized, a much larger porportion of the cases of gastrointestinal and nutritional disturbances will be found to be due to other causes than rhinopharyngeal infection.

Not all infants suffering from ear and throat infections show the same symptomatology. Some infants simply fail to gain in weight but have neither fever nor diarrhea; the throat may be slightly reddened and the ear drums lacking in luster, but not bulging. The blood leucocyte count in those cases is usually elevated. Removal of adenoids and tonsils or paracentesis and possibly antrotomy very often causes a change in the whole clinical picture. Some infants with persistent pyuria show no improvement under the usual methods of treatment until coexistent infections in the ears, nose and throat are cleared up.

Considerable harm would undoubtedly be done if infants' mastoids were promiscuously opened for the treatment of diarrhea and nutritional disturbances without considering the possibility of other causes of the condition. More harm, however, has been done and is being done by permitting unrecognized infection to escape attention. In the light of our present knowledge, treatment of the ears, nose and throat of babies assumes at least as great importance as modification of the diet.

Beyond the age of infancy, infections in the ear, nose and throat do not so often lead to gastrointestinal disturbances, but such infections are often an important factor in bringing about a condition of malnutrition. There are also certain more or less clear cut general conditions of older children which are caused or aggravated by infections in the rhinopharynx or nasal accessory sinuses.

A clinical picture frequently seen is that of a pale, languid, undernourished child, with an occasional hacking cough, and slight evening temperature. Physical examination reveals nothing in the chest, but the X-ray shows enlarged hilus shadows and peribronchial thickening, often extending well out into the parenchyma of the lungs. Small dense areas, such as are usually interpreted as shadows of calcified glands, may be seen. There may be slight functional murmurs in the heart, but no more definite evidence of cardiac disease. The cervical glands in both the anterior and posterior triangles are palpable. The tonsils, if they have not already been removed, are likely to be the seat of the chronic inflammation, and there is a granular pharyngitis. There are often nasal findings indicative of some degree of sinusitis, although this may not be marked. Removal of the tonsils and adenoids results, in a certain number of instances, in general improvement of the patient, and the disappearance of the signs of rhinopharyngeal infection. In others, however, the symptoms persist. Tuberculosis is often suspected, but may be ruled out by negative intradermal tuberculin tests. Roentgenograms of the nasal accessory sinuses sometimes reveal a definite clouding of one or more of the sinuses, most likely one of the maxillary antra. In other instances, roentgenograms after the introduction of lipiodol into the antra may show a definite thickening of the lining mucous membrane. In those cases in which definite sinus in-

fection is found, treatment designed to clear up the sinus infections not infrequently leads to considerable improvement in the child's general condition and the disappearance of many of the symptoms. In the case of malnourished children, it is often difficult to tell how much the rhinopharyngeal infection observed is the result of the poor nutritional condition and how much the cause. Practically all malnourished children, living in cities in the soft coal belt, show some slight nasal signs which may be interpreted as evidence of sinus infection. These findings, however, should not be considered indications for more than the mildest local treatment and the institution of general hygienic measures. Most important is the building up of the resistance by adequate nutrition, and especially the administration of food containing the "A" vitamine, namely, butter, milk and cod liver oil. An abundance of fresh air, free from smoke, is almost essential. Exposure to sunlight out of doors seems an aid, but exposure to ultraviolet light indoors is of very questionable value. When there is definite pus in one of the antra, irrigation and possibly ventilation of the sinus is indicated and is often followed by good results, though reinfection is common. Local treatment of the nasal mucosa with antiseptics and astringents is of benefit. Keeping the nose clean is a very important part of the treatment, and this can be accomplished by irrigations with a weakly alkaline salt solution, or better with a 10 per cent solution of glucose (cerelose). It is our strong belief that radical operations on the sinuses of these undernourished children should not be attempted except for the most urgent indications.

Some children develop periodically symptoms which may be difficult to distinguish from those of appendicitis. They have abdominal pain and at times vomiting. These attacks are often seen in children who are susceptible to repeated nose and throat infections, and the acute symptoms coincide with fresh infections in the tonsils, adenoids and sinuses, and subside with the subsidence of the infection. Brennemann⁶ has especially drawn attention to this syndrome.

A rather frequent accompaniment of chronic rhinopharyngeal infection in children is a condition of disturbed digestion associated with hypochlorhydria.

Asthmatic symptoms are frequently observed in children suffering from chronic purulent sinusitis, although such infections account for only a certain porportion of cases of asthma. When the asthma is due primarily to rhinopharyngeal infection, there is likely to be an accompanying profuse mucopurulent discharge and definite evidences of sinusitis. In asthma due to sensitization the nasal mucosa becomes thickened and edematous, and this undoubtedly predisposes to sinus infection. Roentgenograms in these cases are often misleading, as clouding of the sinuses may occur, due to the swelling of the mucosa. In such cases very little material is obtained on irrigation of the antra, and such cells as are present are likely to be eosinophiles unless an acute purulent rhinopharyngitis is superimposed. In the cases of asthma due to rhinopharyngeal infection, roentgenograms of the chest generally show very marked hilus gland enlargement and peribronchial thickening; in chronic cases some bronchiectasis is commonly present. In these cases, very little improvement in the asthmatic symptoms can be expected unless the rhinopharyngeal infection is cleared up. Unfortunately, in many cases it is difficult to eradicate these infections, but when this is possible very marked improvement in the asthmatic symptoms occurs.

Chronic arthritis has very generally been believed to be one of the manifestations of focal infection. The infection may be in a variety of locations. We have seen three instances in children in which there was definite evidence of infection in the paranasal sinuses. In two of these improvement and ultimately complete disappearance of the acute symptoms of arthritis occurred subsequent to adequate treatment of the nasal sinuses.

The relationship of nose and throat infections to nephritis had been much discussed in recent years. In children the various forms of nephritis may be seen in the early stages, and it is easier to determine possible causative factors than in the case of adults in whom the disease picture is more complicated.

The most common type of nephritis seen in children is acute hemorrhagic or glomerular nephritis. This almost always follows an acute streptococcus infection, generally of the nose

and throat. In the majority of instances the condition subsides after a period of time. Albumin and blood disappear from the urine and apparent complete recovery follows. In a small proportion of cases the symptoms persist, the urine continues to show albumin and a few red blood cells, the blood pressure rises and there develops finally the picture of chronic nephritis with hypertension and renal insufficiency. Longcope⁷ has recently pointed out the relationship between persistence of streptococcus infections in the nose and throat and the appearance of hematuria. He found streptococci in the noses and throats of 83 per cent of patients who had, from time to time, recurrences of blood in the urine, whereas 90 per cent of the patients who no longer showed hematuria were free from streptococcus infections in the nose and throat. Our own experience coincides closely with that of Longcope.

In the light of these observations, it would seem rational to attempt to eliminate streptococcus infections from the nose and throat in cases of persistent hemorrhagic nephritis. This we have attempted to do in a number of our cases, but the end results have not been any too encouraging. In a few patients who have showed hematuria over a period of weeks, removal of the tonsils and adenoids or treatment of the sinuses has been followed by complete disappearance of blood and albumin from the urine. Frequently when operations are done on the noses and throats of these patients there occurs immediately after the operation an exacerbation of all the symptoms, which may then be followed by recession. It is our feeling that nothing in an operative way should be attempted until the patient has been observed for a reasonable time to determine whether or not spontaneous cure is likely to occur. On the other hand, in the case of children who have suffered from glomerular nephritis for longer than six months, we have not seen any who have finally become free from evidences of nephritis. In some children, treatment designed to remove infection from the nose and throat seems to have resulted in a slower progression of the nephritis to its fatal termination.

A second form of nephritis seen during childhood is parenchymatous nephritis or *nephrosis* characterized by marked edema, oliguria and albuminuria, but without hematuria, hyper-

tension or nitrogen retention. This condition at times develops without assignable cause, but in a very considerable number of cases is preceded by a rhinopharyngeal infection, such as a cold in the head, followed by a purulent nasal discharge. In other cases the nephrosis has been observed to develop after osteomyelitis, pulmonary infections, empyema thoracis or abscesses. Once the condition of nephrosis has developed, there may be marked remissions and exacerbations, the latter often being coincident with fresh infections, particularly in the nose and throat. This has led to the suggestion that nephrosis is caused primarily by infection.

Clausen⁸, in 1925, reported from this clinic a series of eleven cases of nephrosis, in all of whom rhinopharyngeal infection was found to be present. Of these eleven patients four recovered after treatment designed to clear up infection in the nasal accessory sinuses. The infecting organism was most frequently a staphylococcus. Aldrich,⁹ in 1926, reported on nineteen cases of nephrosis with edema, in all of whom there was evidence of definite nasal infection. Successful drainage of abscesses resulting from these infections gave constant and promptly beneficial results. Many of the exacerbations of edema subsided, with spontaneous improvement in the nasal infection. The organisms found included streptococci as well as staphylococci. In this series of cases treatment by diuretics, dietary regimens, and dehydrating measures combined with fluid restriction resulted in no usual or constant improvement in the symptoms. Mitchell¹⁰ observed the frequent association between nasal sinus disease and nephrosis. He reports in detail an illustrative case in which drainage of an antrum resulted in disappearance of the symptoms of nephrosis. The organism most frequently found in his series of cases was a staphylococcus.

Franconi¹¹ and Nourse¹² have each reported cases of nephrosis which recovered following the drainage of pneumococcus abscesses in the abdomen.

Davison and Salinger¹³ reported on twenty-six cases of nephrosis admitted to a hospital during the past fifteen years. Five of these who were carefully examined had clouding of one or more sinuses; irrigation performed on three of these

revealed no pus, and in two there was no diminution in the edema following irrigation. In one case irrigation seemed to affect the edema favorably. Only six of the cases gave a history of an infection immediately preceding the symptoms of nephrosis.

Since Clausen's report in 1925, there have been admitted to the St. Louis Children's Hospital ten typical cases of nephrosis. In seven of these cases there was present a purulent nasal discharge; in two there was a chronic pulmonary process, and in the remaining one no evidence of infection was demonstrated. The sinuses showed clouding by X-ray, but the patient died before an intranasal examination was made. Two of the patients with evidence of rhinopharyngeal infection recovered without treatment and at present show no edema or albuminuria. Of the patients who received no intranasal treatment four died of streptococcus or pneumococcus peritonitis. One patient with sinusitis had radical operations on all of the sinuses. His edema disappeared after the first drainage operation, although the urine never became entirely free of albumin. He ultimately died of streptococcus peritonitis following shortly after a sinus operation. One patient with pus in the maxillary antra was improving spontaneously at the time windows were cut in each maxillary antrum. He became free from edema and has shown neither edema nor albuminuria for the past two years.

The question as to whether or not infection is the primary cause of nephrosis cannot be settled on the basis of available evidence. Patients with nephrosis are known to be very susceptible to infections of all sorts, and such patients would be particularly liable to develop nose and throat infections during the course of the disease, even were these infections not originally present. The fact that clinical improvement often occurs coincident with the spontaneous clearing up of the infection or as the result of drainage of infected areas may merely mean that the infection has caused exacerbation of a condition already present. If nasal accessory sinus disease leads to nephrosis, it is difficult to explain why nephrosis is a very infrequent manifestation of sinus infection. Certainly many individuals with severe sinus disease never show the least evidence of

nephrosis. There may be a constitutional factor involved. Such variability in the results of infection in different individuals is not uncommon. It is a well known fact that relatively few patients develop nephritis after scarlet fever, yet no one questions the fact that scarlet fever is the etiologic factor in post-scarlatinal nephritis.

In some instances the evidences of nephrosis disappear after drainage and ventilation of sinuses, yet active nasal infection may still be present. This suggests the possibility that the growth of certain organisms under the anaerobic conditions existing in closed sinuses may result in the production of a different type of reaction than when growing under the aerobic conditions induced by operation. It has not as yet been possible to reproduce nephrosis in animals by infecting them with the organisms isolated from the sinuses of patients with nephrosis.

Leaving aside the question of the etiology of nephrosis, the fact remains that a fair number of these patients do show rhinopharyngeal infections and that the treatment of these infections is often followed by improvement in the clinical condition. It is our feeling that in all cases of nephrosis a careful examination of the throat and nasal accessory sinuses should be made and that when definite infection is found it should be appropriately treated as in the case of any other disease. The fact must not be lost sight of, however, that these patients have very little resistance to infection and that radical operations on the nose and throat are occasionally followed by a streptococcus septicemia, peritonitis and death. On the other hand, many of these patients die from septicemia or peritonitis in whom no treatment has been applied to the nose or throat.

There are other conditions in children which seem to be caused or aggravated by sinus infection, but the ones mentioned are those most frequently observed and in which the influence of the infections seems most pronounced.

In any individual case it is important that the influence of sinus infection should be properly evaluated—it may be an incidental finding or it may be the one factor which retards or prevents recovery. To treat the one radically may be an error; to neglect the other an equally great error.

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V.

PAGES FROM THE ANCIENT HISTORY OF BACTERIAL INFECTION THROUGH MUCOUS MEMBRANES.

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It must have been many years before this journal made its first bow to the medical public that one who contributed to some of its earlier numbers wrote elsewhere:* "The question of immunity and recovery from disease has occupied the minds of medical men ever since the dawn of history. In one form or another it has been recognized that there are two opposing forces at work in any case of disease—the malignant tendency of the malady and the resisting power of the victim's organism. Even the most savage and uncivilized races seem to have a glimmer of this clinical fact."* This very generalized and quite trite exposition written in 1890 it would be difficult to refute, but, elementary as it may seem, it is a conception every one dealing with disease must ever have clearly in mind. Now, nearly forty years later, the writer, who had sufficient cause then to do so, but now that he is old might find still further reason to amplify the statement if he attempted to catalogue, so far as he could, all the factors that are "malignant" and all that are the "resisting" ones,—in tuberculosis, for instance. Anyone thinking to do that, after these all but forty years, would have a large contract on his hands. He would find them much interlaced, some factors that are malignant in one case are not malignant in another, some that are resisting are sometimes predisposing factors, but I cannot find space here even so much as to exemplify the old adage that what is food for one man is poison for another, for this essay is to be an his-

*Wright, Jonathan: The New York Medical Journal, May 3, 1890.

torical one in honor of Dr. Loeb's memory, who, as editor and founder of this journal, did so much to advance the fertility and the beneficial influences of the fields in medicine it has cultivated.

As to tuberculosis, even back in those days it was known that six people out of seven who have had tuberculosis in their bodies do not die of tuberculosis. We have as a profession moved to the point, which was even then quite plain to some, that the tubercle bacillus is a part of the process, but no more and no less the cause of tuberculosis than the skin is the cause of dermatitis. Without the tubercle bacillus one does not have tuberculosis, and without one's skin—I need not waste space on that. It is one of the factors in the etiology, but it is a part of the process. Such was, more or less vaguely but insistently expressed, the standpoint of the author of the paper just referred to, but in it, too, it is stated that the intact mucous membranes of the upper air passages offer great resistance to the entrance of the organisms in constant contact with them.

Abrasions of the buccal mucosa are mentioned, but the tooth brush as the cause of them is not. Indeed the conception of the mechanism whereby infection passes through the first barriers to infection was not, at the time this paper was written, at all a part of the exposition of infection of the upper air passages, and the sanitarians much more gaily then than now recommended, as one way to prevent infection, scraping epithelium off the gums, but the tooth brush is still devoutly a part of the education for public health. From the teeth and their roots and the surfaces it was tacitly accepted that infection must enter, and from the tonsils, too, but no question was raised as to how its agents entered. On the contrary, interest was focused at an early date on the lymph glands as bacterial filters after they had entered. It was not because it was on the surface, it was because it was a lymph gland that the tonsil became an interesting question, first in the discussion, and only after the mechanism of internal membrane physics became intensely studied that tonsillar external surfaces with bacteria against them claimed an attention which they have not yet sufficiently received.

The lymphatic glands and the network of the lymph spaces and the progress of bacteria along them were studied in this

preserologic era of bacteriology, but the network of the surface cells shared no recognition as the first and, on the whole, as the most important bulwark of defense against infection, and as such they are not studied today. It was not long, however, before the downfall of Koch's postulates moved out of our way one obstruction to the progress of thought as to infection. I find them quoted with derision in another old paper* some five years later. They were:

1. In every disease due to microorganisms, the offending etiologic organism is always present in that disease.
2. It was never found in man without the disease. By this time (1895) the falsity of this statement was notorious.
3. Inoculation with it always caused the disease, but exceptions to this began to be noticed.

The selection of etiology as the basis of the classification of disease after the advent in medicine of bacteriology was an undoubted improvement on what had obtained before, but the bacterium as an exclusive factor in the etiology of even bacterial disease seems every day less enlightened as our knowledge of the laws of nature progresses, yet despite this it seemed a great gain over clinical differentiation. Like all classifications in medicine it soon "wrought some evil in crystallizing a certain order of mental process into a form of intellectual non-receptivity which renders further advance in individual cases impossible."

In the thirty years or more which have elapsed, the high contagionists have passed from troubling the serenity of young men with their faces set toward the future who have since had troubles of their own. Already in laryngology, more specifically in stomatology, we had no reason in studying the etiology to seek for a solution of continuity in the mucous membranes of the upper air passages. "An open wound is not necessary for penetration, since the bacilli can find an entrance through the epithelium." In the face of this and of the fact which bacteriologic investigators had long since shown—that all sorts of bacteria, pathogenic as well as nonpathogenic—are in con-

*New York Medical Journal, September 21, 1895.

stant contact with the epithelium of the tonsillar crypts* and there as well as elsewhere on the surface of mucous membranes the tubercle bacillus, of which there are many strains and various races, "though being the *sine qua non* of tuberculosis is, after all, practically, especially from a prophylactic or hygienic point of view, a minor element in its multitudinous factors."

It is quite apparent then that as long ago as 1895 the author of that paper and many other workers had the question postulated by undeniable facts: "What is it allows the bacteria to penetrate sometimes in some conditions and not at other times or in other conditions, which usually for long periods of time, usually in fact forever, keeps them out?" Though I do not find he uses exactly those words, that was the problem, stated as succinctly as possible, with which he was confronted before the rise of intense laboratory interest in serology diverted attention from the first line of defense in immunity. In a way, and possibly in a useful way, as it has turned out, a "red herring was dragged across the trail." Such questions and such ways of evading or missing them nature has and is, as history teaches us, ever ready to present and to withdraw from answering except in an abortive way. How Galen escaped discovering the way the blood actually does circulate I never fully understood. It is to be constantly found in history, however, that men have often, ever staring them in the face for centuries, ever presented to the mind of reasonable men, not questions and problems simply, but facts unobserved.† No historian of thought, no psychologist has ever explained this satisfactorily. They

*Of late years with thoughts frequently occupied with the conception of the mutation of nonpathogenic bacteria it has repeatedly occurred to me, from the drift of research and its literature, that it is par excellence within the tonsillar crypt that the bacteria, all of them immortal, have there learned to put on pathogenicity and lay off nonpathogenicity. Slaying an epithelial or wandering cell may tell decidedly for the survival of a race of bacteria. A study of the forms usually more or less benign of actinomyces awakened that surmise in my mind many years ago. American Journal of the Medical Sciences, July, 1904.

†"Thoughts lie dormant for ages; and then, almost suddenly, as it were, mankind finds that they have embodied themselves in institutions." (Whitehead.)

give no heed to such facts whatever until suddenly some common, ordinary man like Harvey, who erred as much as he grasped the truth, calls men's attention to what had always been plain as a pikestaff. There is a good deal of nonsense talked about master minds.

Prudden had shown that he could produce a lesion identical with tubercle without the tubercle bacillus and helped the postulates of Koch, his old teacher, over the rim of the horizon, and a French author had said inversely, "the bacillus is not enough, we must have the characteristic lesion—the tubercle"—to make a diagnosis, and it might have been added, even having both is not enough. Neither tubercle nor the bacillus is a disease; a disease is the reaction between them, and the animal organism in which they for the time lie, which makes the concept of tuberculosis. A bacillus and a tubercle are material facts; a diagnosis is a concept.

I am free to say I did not have all this precision of thought so well worked out, but philosophers and wiser men have taught me since my youth that a keener analysis by purely dialectic methods than now obtains in science would frequently not only clarify the thought of young and old but very much aid research itself. A man may have the tubercle and the tubercle bacillus too in his body the whole of a long life and never have a sick day—not necessarily even a diseased imagination. This paper, in 1895, nevertheless asks the question, after enumerating the clear evidences of the existence on the surface of mucous membranes of forces resisting the entrance of bacteria, "What are these protective factors in the upper air passages?" Eugen Fraenkel and Mr. Lake had already demonstrated the existence of tubercle bacilli and pyogenic cocci in and between the epithelial cells, and it was Friedrich's idea that the pneumogastric, "in some cases of pulmonary phthisis, by becoming affected produces a trophic lesion in the larynx which allows the tubercle bacilli to enter." It was, however, not the belief of the author of this 1895 paper that any lesion, either trophic or any other, was necessary to admit it, but the idea of the intervention of the vagus nerve, or some other nerve perhaps, thus took root in the author's mind.

The matter of this paper received favorable comment in the editorial columns of medical publications, but neither the matter nor the reaction to it, nor similar expressions of opinion from other observers produced any reaction at all upon laboratory workers. A year later there was, in 1896, a further report made before the American Laryngological Association, illustrated by a colored drawing (Plate I) made from "sections of papillomatous tissue removed from a tubercular larynx in a patient far advanced in pulmonary and laryngeal tuberculosis," showing a stream of tubercle bacilli passing through the epithelial surfaces. The picture was a striking one and illustrated how apparently a pathway broad at the surface but narrowing as it descended was paved with the fuchsin stained bacilli, where the resistance had ceased, to their entrance, at one place only, from the surface to the tissues below. It impressed the observer very strongly that in all the sections it was only at this point their entrance could be noted—only in this limited area could it be seen—a stream converging from the superficial cells to the lymph spaces below the epithelial line. Conclusions drawn from tinctorial reactions, especially the stain of waxy coated organisms, nearly always need other support, but that existed in the general conception.*

The demonstration of living virulent microorganisms of various diseases on the different mucosæ of healthy people became at this time very frequent in medical publications. "Many people with normal upper respiratory passages are found to have in the secretions the diphtheria bacillus of Löffler," and that is noted "as one of a series of observations which is slowly tending to modify our earlier conceptions of the relative importance of bacterial influences in the etiology of diseases with which they are associated." Dr. Park and I published a paper in 1895† showing the mechanical factors taking part in the elimination of bacteria from the air current of the upper channels of the nose were gravity, filtering action of vibrissæ in the nasal vestibule, the wash of sterile secretion from glands in the region not reached by the inmoving air current, and that

*New York Medical Journal, September 26, 1896. Transactions of the American Laryngological Association, 1896.

†New York Medical Journal, February 5, 1895.

the nasal mucus is a poor culture medium. It had been noted previously that the structure and secretions within the nasal cavities removed the vast majority of bacteria before the air reached the larynx.*

"The behavior of the larynx toward tuberculous infection has long been an interesting puzzle to laryngologists. I have long believed that its careful study would elicit important hints as to the mode of tuberculous infection. . . . One very great stumbling block in the discussion of the means by which the larynx becomes tuberculous is contained in the terms of the problem itself as usually expressed. 'What is the cause of tuberculous laryngitis?' It would be better to put the question thus: 'Why is it that the larynx usually escapes infection with the tubercle bacillus?' "† When the sputum laden with bacilli from tuberculous lungs is constantly passing over and clinging to the surface epithelium, since we know the bacilli can pass at times, it was easily to be inferred there was something prevented them passing at other times. There had been a good deal of evidence brought forward by Cornet,‡ the high priest of the high contagionists, to suggest that it was rather by the inhalation of dried and powdered sputum that the tubercle bacillus found its way to the mucous surfaces of man's respiratory passages than by the inhalation of globules of fresh sputum containing bacilli coughed into the air. Various considerations were offered§ to invalidate this view. He had shown that some absorption takes place "when various powdered substances are blown upon the surfaces of mucous membranes," but it was urged that this was not sufficient reason to conclude that bacteria were absorbed in this manner. The rubbing of streptococci on the mucosæ of animals had given positive results, but again the technic was criticised as defective in demonstrating how infection occurred practically in man. Goodale|| and Hendelsohn¶ had introduced carmin granules in the crypts

*New York Medical Journal, July 27, 1889.

†New York Medical Journal, June 24, 1899.

‡Berliner klinische Wochenschrift, No. 2:12, 1899.

§New York Medical Journal, April 7, 1900.

||Goodale: Archiv für Laryngologie und Rhinologie, 1897, VII, p. 90.

¶Hendelsohn: Archiv für Laryngologie und Rhinologie, 1898, VIII, p. 476.

of the tonsils, and Wright had repeated their work, but again the conclusions rested on an insecure basis. None realized then that the same surface tension does not exist between the cells and dust as between those and live bacilli on the surface, "for the dead are but dust," a little different from carmin granules, perhaps, in the dynamic reactions between surfaces, but different from live bacteria.

Much was gained by realizing this, but this was yet in the future in 1901, when it was said: "The problem which now confronts us in the study of the causes of tuberculosis is the necessity of finding a method by which we may investigate those factors which render the morbid influence of the tubercle bacillus an efficient one in the production of disease in the animal economy of its host. . . . The mucous membranes of the upper air passages not only receive the primary deposit of the infective agent, but they are subsequently, in case of the establishment of a pulmonary nidus, washed by the virulent cultures thrown off from the distant point of development without themselves, as I have just said, in the majority of cases, presenting evidences of morbid action. . . . Shall we say that this greater exposure has in the process of evolution produced a graded local immunity which is inherited?" It was becoming evident that we must think in terms of evolution, not of the bacillus alone from a possibly nonpathogenic strain of its species, but of an immunity to it acquired by its animal host. This paper,* from which these excerpts are taken, was a review of the problems of tuberculosis of the upper air passages, read before the New York State Medical Association in 1900, and many others of like tenor were presented by the author at the New York Academy of Medicine, the American Laryngological Association and elsewhere. There was at that time (1901) beginning to be manifested some, though very feeble, interest in surface infection. Laboratory research was then beginning the great development of the subject of the internal defenses of the organism—the serology which dominated the interest of bacteriologists for twenty years. Much of it hinged around tuberculosis.

*The Medical News, New York, January 19, 1901.

I have referred to the attempt made in 1889 to ascertain if bacteria in the inspired air usually reached the lungs or rather the ultimate bronchioles. It was pretty well proven that they did not. Indeed, considering the level of the residual air, it was all but certain *a priori* that they did not. In Virchow's Archiv.* Dr. Saenger, some fifteen years later, asked and answered that question pretty conclusively—that neither they nor dust could do so. They must arrive by other channels. Repeatedly I had found carmin granules in the epithelia of the lung vesicles of rabbits after injecting emulsions of it in the hind leg as well as in the tonsillar region. Indeed, I found them in the tonsil itself after injection in distant areas. From this time it was pretty generally believed by the profession at large, though some had entertained that opinion long before it, that infection directly by the lungs was no longer a defensible theory. Though the food passes through the pharynx as well as the air, the latter for many became the chief carrier of infection, and as the acid reaction of the stomach resisted or destroyed many germs this seemed not a promising entry port. Still no one joined in the interest as to the question of how a bacterium passes the surface epithelium in such a manner that it becomes an enemy within the gates, to be dealt with by the internal defenders. All interest was absorbed by the enticing wonders and complexities of serology. Ehrlich's symbols suffused the world of science to the exclusion of all other interests in laboratory work. Like sheep, one at a time and over the fence once, all workers were huddled into one corner.

By 1903 von Behring† had declared that the tubercle bacillus enters everybody's organism in infancy, and Naegeli long before this had furnished a basis for the assertion by pointing to postmortem reports, made more carefully than before Koch's great discovery. It was proved that practically all dying beyond thirty or forty years of age present in their bodies evidence of having been at some period of their lives the hosts of the tubercle bacillus‡ which had formed tubercles there,

*No. 167, Heft I.

†Deutsche medizinische Wochenschrift, September 24, 1903.

‡New York Medical Journal, April 2, 1904.

and von Behring believed it had entered with the food, as a rule, but by what mechanism it crossed the epithelial barrier and by what it was excluded nobody thought to inquire, although dust, as had always been known from the anthracosis of coal miners, easily passed, and the inference from high contagionist theories, which then reigned, was unavoidable that bacteria could but rarely pass. There was no need of going to a laboratory with such undoubted facts outside of it and such false but widely held theory within. (Fig. 3.)

In a case of supposed actinomycosis of the tonsils,* in which the organism was probably one of the numerous benign strains but had undoubtedly passed the borderline of the epithelium and was having a marked effect on the cellular metabolism and causing its proliferation (Fig. 4), the query arose†, "Could the organism under such circumstances pass through the intact squamous epithelium?" At one place it seemed plainly growing in the connective tissue beneath (Fig. 5), but that it existed and was growing among the epithelial cells there could be no doubt. As organisms of this kind and sometimes referred to as false actinomyces are frequently seen in tonsillar crypts among the detritus of an hypertrophied and desquamated epithelium the suggestion was insistent, but not thoroughly heeded, that the tonsillar structure existing at the crossing of the pathways of food and air possess a mechanism of some kind which, except on rare occasions and very sparingly then, allows them to pass. Tonsils from Pittsburgh were examined, and coal dust was seen to be passing readily from the tonsillar crypts just as the carmin granules did in the experiments of Hendelsohn, Goodale and Wright. *"It seems, therefore, that the dust passes, but cocci and bacilli of the same or smaller dimensions do not pass into (the structure of) the quiescent tonsil."* I italicize in my quotation here this sentence. The italics were not used then, but the sentence marks a turn in the mind of the writer toward an explanation which later led to much further work on his part and still further to that

*I was indebted to Dr. Lecount of Chicago for photographs which are now published for the first time.

†American Journal of the Medical Sciences, July, 1904.

of Dr. Stuart Mudd.* Manifestly the dust is dead and the bacterium alive, with all the potentialities of life on the surface, and so is the surface epithelium, and evolution has played its part in the relationship between them. Some change wrought by the actinomyces and the other more common forms of bacteria lying regularly against it occasionally opens the door in the cryptal epithelium.

The glimmer of this, which is now so easily formulated, was only just becoming apparent then. There are many kinds of bacteria, and of actinomyces among them, which probably do not alter the forms of the surface epithelium, but some evidently do. Many tonsils present striking features of keratosis of the epithelium of their crypts. Something makes such cryptal lining differ thus widely from that of other tonsils. It may not be the character of special bacteria that lie in certain crypts, but we are privileged to infer that it is, for no one has ever taken the trouble, so far as I know, even to attempt to give a reasonable explanation of keratosis of the tonsils. It just lies there for every microscopist to see and no one to no-

*I take this opportunity of referring to the very valuable work of Dr. Mudd, who became interested in the matter now a number of years ago, and more recently has begun to publish the results of his laboratory work at the Rockefeller Institute and elsewhere. It was involved in such highly technical detail of bioelectrophysics I have not been able to summarize it here. It is largely confirmatory of the conception here reviewed, however. Out of a very much longer list of papers, all of which have an important bearing on it, I may mention the following from the papers of Dr. Stuart Mudd:

The Penetration of Bacteria Through Capillary Spaces. I. Motility and Size as Influencing Filterability Through Barkefeld Candles. *Journal of Bacteriology*, VIII, 459, 1923.

The Penetration of Bacteria Through Capillary Spaces. II. Migration Through Sand. *Journal of Bacteriology*, IX, 143, 1924.

The Penetration of Bacteria Through Capillary Spaces. III. Transport Through Berkefeld Filters by Electroendosmotic Streaming. *Journal of Bacteriology*, IX, 151, 1924.

The Penetration of Bacteria Through Capillary Spaces. IV. A Kinetic Mechanism in Interfaces. *Journal of Experimental Medicine*, XL, 633, 1924.

Certain Interfacial Tension Relations and the Behavior of Bacteria in Films. *Journal of Experimental Medicine*, XL, 647, 1924.

On the Surface Composition of Normal and Sensitized Mammalian Blood Cells. *Journal of Experimental Medicine*, XLIII, 127, 1926.

tice so far as to be interested in the etiology of a very striking phenomenon. I suppose most observers, if they think of it at all, figure it as irritation "like a corn" and let it go at that.

"From the experiments of Goodale and others with colored granules, from my own observations of dust particles passing the epithelial layers in health and of *bacteria passing the epithelial layer in disease*, it is evident enough that there must be something beyond mechanical obstruction which under ordinary conditions of health keeps the tissue beneath the epithelium free of bacterial life which swarms in some of the crypts on the outside of the epithelial cells. . . . At the forks where the foodway and breathway meet we have a local phenomenon which puzzles the clinician and the microscopist, and if it leaves the laboratory bacteriologist complacent it is only because of his limitations." As we have seen, in slightly different terminology, the writer had already suggested that it "is some sensitive mechanical arrangement which closes the stomata and interstices of cells to the ingress of bacteria and allows the inorganic particles to pass through, but perhaps this does not help the matter very much."* It certainly was a very vague adumbration of a physical phenomenon, but it was very helpful in orienting the writer's mind in the right direction. The problem was looked upon from the ancient standpoint of Alcmaeon. The tonsillar crypt in health was the seat of an equilibrium between infection and immunity. The next year (1906) he wrote further in illustration (Plate II) of "the difference in the behavior of dust from that of bacteria in the tonsillar crypts."† I have omitted to say that Pirera‡ had in 1900 written in support of the statement, which even then had been previously made, that dust frequently and readily passes through the epithelium of the tonsillar crypt, but his technic met with much criticism, and it certainly led him greatly into exaggeration.

It had become necessary to conjecture or rather to form some idea of how any mechanism, physical or biologic, could have been set up there in animal evolution, and a considerable

*The Medical News, March 4, 1905.

†New York Medical Journal, January 6, 1906.

‡Archivo de Laryngología, Aprile, 1900.

time was spent in discussing the question of the heredity of organic and inorganic matter. To this I have no room to refer further than the footnote permits.*

Referring, however, to the 1906 paper on the equilibrium in the tonsillar crypt, the author makes the remark, "there is good presumptive evidence that pathogenic bacteria, which in a state of equilibrium are harmless inhabitants of the tonsillar crypts are under certain conditions absorbed through tonsillar epithelium," and he gives a short resumé of the experimental evidence, the inferences from which have been here given more in extenso, and he goes on to say: "The shock of a bone fracture, the nerve collapse accompanying a profuse hemorrhage, even the small shock of a nasal operation, the irritation of uric acid, the shock of cold to the surface, may apply the nerve stimulus necessary to open the portals to an infection of osteomyelitis, profound sepsis, perhaps rheumatism or a cold in the head, but man or beast does not practically acquire these affections or their like by having a laboratory practitioner blow in his throat a pure streptococcus culture. He cannot artificially produce that concatenation of circumstances which forms the practical etiology of nature. The phenomena following such an experiment may be said to be chiefly of academic or rather of laboratory interest."

In this paper the writer draws attention to Wilson's† conjecture that the kinesis of cell division presents phenomena similar to the arrangement of iron filings in the field of a horseshoe magnet, and in the problem of the bacterium in the tonsillar crypt he makes the conjecture of a similar reaction "between the protoplasm of the epithelial cell and that of the bacterium." He found such suggestion, too, in the gyrations of monocellular organisms around one another in a hanging

*The Origin and Heredity of Matter. A Critical Review of the Literature. St. Louis Medical Review, August 4, 11, 18, 25; September 8, 15, 22, 29, 1906.

The Heredity of Form as Illustrated in Pathology by a Study of Cysts of the Middle Turbinate Bone. American Journal of Medical Sciences, May, 1907.

The Theories and Problems of Heredity. New York Medical Journal, January 9, February 13, April 3, 24, May 29, 1909.

†The Cell in Development and Inheritance. By Edmund B. Wilson. 2nd Edition. Macmillan, New York, 1904.

drop under the microscope. Dry bran or thin camphor shavings on water also represent the motions of inanimate particles to the adjustments of surface tension. The conception of Bordet and others of there being a negative and a positive "chemotaxis" taking place between a bacterium and the surface of its host's internal lymph spaces and other passages is utilized in illustrating "the relationship of the epithelial lining of the tonsillar crypt to its bacterial contents," and he points to the probably fundamental relationship to the more invisible reactions of serology.

In pairs of tonsils to be removed the crypts of one of them in each case were curetted a few days or a week before the operation; in some long, deep cuts were made, one tonsil always serving as control. In one of several pairs deep punctures were made with a sterile probe from the bottom of the crypts to carry some of the cryptal bacteria far beneath the epithelium. These latter only gave positive results. At the bottom of the probe puncture in some cases and along its lateral walls near the bottom, in marked contrast to the more superficial levels, small colonies of cocci could be seen, which had been growing in the sections of the hardened tissue. Bacterolysis had probably destroyed the bacteria in the curetted crypts, as they were indistinguishable in the detritus found there. Evidently this was the only suggestion to be gained from this series of experiments, but in this paper is given a colored plate (Plate II) of a section of tonsil, dusted with carmin granules before operation, and in places are plainly shown the granules streaming past bacteria covered epithelium deeply into the cellular spaces of the connective tissue. Certainly no more striking exemplification could be given of the long surmised fact that the mechanism at the surface is a selective mechanism, which allows harmless inorganic matter to pass and keeps out possibly dangerous protoplasm. It was now more than a surmise that this selection, this differentiation, was due to the variation in the indices of surface tension, respectively, between dust and the cells and between bacteria and the cells.

By this time it had become apparent that "we must go back to the fundamental attributes of atoms and molecules for an understanding of many of the phenomena of immunity," not only of those of serology, but for those of the external barriers

of disease represented by the epithelium of the mucosæ. In these days the conception has extended so far in this direction that I am reading in a French publication* not only of the living molecule and atom but of the living electron, and elsewhere I have seen talk of dust falling on the electrons. This order of ideas is broached in another paper† of about the same date I have just been drawing upon, and the title of it is "The Primordial Nature of the Forces Exerted Against the Penetration of Bacteria Beneath the Surface of the Body." "Haeckel and the ancient philosophers of Greece, thousands of years before him, had conceived of sensation as the manifestation in animals endowed with consciousness of the law of molecular attraction and repulsion." In 1906 recent experiments in physics had been promulgated which have finally led to an overturn which has confused and humiliated the theories of physics hitherto entertained, but they had unveiled other manifestations of more direct application here. In cell division we had become familiar with the manifestations of intracellular electrodynamic polarity. It was but a step to apply the principles to the external relations of cell and bacterium. The work of Calkins‡ in 1901 on the protozoa, supplemented by those of Loeb§ and Jennings|| in 1906, were most suggestive. The phenomena described by these investigators threw a flood of light over the birth and behavior of all living cells, with that of Wilson already referred to, and their relationship to our problem was frequently discussed in the papers of this period, but though they were utilized also for the light they threw on the heredity of living matter, it was plain, as I have intimated, that the search must be carried into molecular forces.

"In attempting to identify the histologic changes in the lymphoid tissue of the pharynx which are coincident with its regression in adolescence, I have run foul . . . of the

*Nodon, A.: *Révue Scientifique*, 1927, No. 20. Les nouvelles radiations ultrapénetrantes et la cellule vitale.

†New York Medical Journal, January 20, 1906.

‡The Protozoa. By Gary N. Calkins. Macmillan Co., 1901.

§The Dynamics of Living Matter. By Jacques Loeb. Columbia Press, 1906.

||The Behavior of the Lower Organisms. By H. S. Jennings. Columbia Press, 1906.

metabolism of proteid into fat (and) of fat absorption at the surface."^{*} At this time some additions to the ordinary technic of the laboratory of practical value had been made in the study of the fats and their derivatives, the lipoids, which were a great advance over the osmic acid stains, by which neutral fat had long since been demonstrated in and about tissue cells. These methods were employed in a study of the tonsillar and other tissues of the upper air passages. It opened a new world to me, and in its light a report was made of it in a series of papers in the *New York Medical Journal* in 1907.[†] The ports of entry, the channels of infection, the electrodynamics of the surfaces of living protoplasm, the nature of colloids, intracellular dynamics, were the headings under which this analysis was made. Reference was made to the principles of physics, which demonstrated that the simple contact of heterogeneous bodies is sufficient to engender electric action, and when the bodies are living bodies it is par excellence the lipoproteids which take part in it.

The conviction became a fixed one that "without them scarcely a single change occurs in cells, and there is no vital reaction between the cells without them, as Le Bon formulates it."[‡]

Sherrington had taught us that "in the limiting layer separating two media forces act which depend on mutual relations between the two media in contact"—not Sherrington alone, but Lillie and many others. These principles had long been known in the world, and their application, borrowed from physics, had been made frequently to many problems in the internal economy of man, but nowhere to those of his mucous surfaces. The phenomena of artificial cells and the experiments of Lippmann had shown how the heredity, so to speak, of inorganic matter was handed over to the organic, for in demonstrating the astonishing shapes and movements of liquid crystals he used those lipoid compounds which had been or were to be found in tonsillar epithelium everywhere and in other cells, but rarely so abundant elsewhere as near the epi-

^{*}New York Medical Journal, December 15, 1906.

[†]February 16; March 9, 27; April 6, 27, 1907.

[‡]Le Bon: *L'Evolution de la matière*, 1905.

thelial cell borders in the pharynx. "With microscopic objects, such as a bacterium in contact with an animal cell in the tonsillar crypt, it is manifestly impossible directly to demonstrate (by the electrometer or microscope) the dynamic relationship between the two, but it is the purpose of these papers* to New York Medical Journal, February 11, 25; April 8, 22; May 13, 1911. collect from near and distant fields enough circumstantial evidence to remove this assumption, from the field of speculation at least."

What had become known of phagocytosis at that time was drawn on as suggestive within the tissue of what occurred at its external boundary, the chemotaxis of monocellular and multicellular organisms, referred to in papers ten years or more earlier—the agglutination and other phenomena of bacteria in serology. In these articles the mechanobiologic standpoint in medical problems was again reviewed, especially as to the part colloid phenomena played in the various processes, where surface tension is the form of force manifested in the surface films of biophysics. Lehmann's work† was utilized in discussing the differentiating power of cells in their reaction to different kinds of particles with which they come in contact in the tonsillar crypts, though the work of American biologists several years earlier, cited here, had anticipated many of the conclusions. As to the tonsillar crypts, "We see them repelling bacterial protoplasm and absorbing inanimate dust." Lehmann says that when fluid crystals are allowed to grow in a solution

*Many have already been cited and abstracts made from them. The titles must serve for others in showing somewhat the drift of investigation and comment.

The Tonsil from an Evolutionary Point of View. The New York Medical Journal, August 8, 1908.

The Mechanobiological Standpoint in Medical Problems. New York Medical Journal, October 10, 15; November 7, 1908.

The Theories and Problems of Heredity. New York Medical Journal, January 9; February 13; April 3, 24, and May 30, 1909.

A Resumé of Some Work on Infection Through the Tonsillar Crypts.

Some Laboratory Notes on the Presence of the Fats and the Lipoids in the Lesion of Rhinoscleroma and on Variations in Its Bacteriology. New York Medical Journal, March 18, 1911.

A Contribution to the Study of Fats and Lipoids in Animal Tissue.

†O. Lehmann: *Physikalische Zeitschrift*, XI, January, 1910.

which holds in suspension particles of a foreign substance, rust, for instance, they appear as clear round bodies in a dark background due to the exertion of repellent surface tension forces between the fluid crystal and the dark granule. Protozoa act in the same way when starch, sand and debris touch them, while they quickly take in organisms, living or dead.

In view of the physical experiments of Lord Rayleigh, which showed that a film of oil six-thousandths of a micromillimeter in thickness on water was enough to stop the gyrations of camphor flakes, as observed on uncoated water, we are led to conjecture that a very minute coating on an epithelial cell of a lipoprotein of proper surface tension suffices to repel a bacterium, and one with a bad index is enough to allow one to enter. A nerve impulse carrying an electric charge, having its origin in some distant area of stomach disturbance or chilling of the surface, or what not, deposits salts on the surface by means of electrolysis, changing the reaction and brings about the entrance of a diphtheria or tubercle bacillus or typhoid germ, or, more commonly perhaps, a streptococcus. This may result in disease, though the organism may have dwelt in a crypt unharmed, but surviving, for years. In a contribution to the study of fats and lipoids in animal tissue, it was shown that the fat and soap and cholesterol and lecithin, which exhibit the properties noted outside the body in artificial cells and liquid crystals, are present in the epithelial layers of the surface of tonsils. The cholesterol being in abundance in quiescent tonsils, in the state of recovery from an inflammation the cells were often found loaded down with soap. The same observation had been made by others in lung inflammations.

It remains to mark a fact repeatedly noticed by me in bacterial stains of tonsillar tissue. Where you find keratosis and transmutation of the cryptal epithelium into the squamous type and one edge of a cell is peeled off and protruding far into the lumen of the crypt, with the other end fast, the free end, which we identify from the way it reacts to stains, is probably "dead." In this state it is permeated freely by bacteria from the crypt, and no bacteria are to be found in or against the attached or "live" end.

There is a short resumé of the work, referred to more at length here, in the textbook on the nose and throat, by Wright and Smith (1914), and the suggestion is made there that inasmuch as Jacques Loeb had elucidated a biochemic mechanism in muscle cells as due to currents of electricity set up, not by contact, but by induction in an exposed nerve, and this results in the formation of or deposit by convection of certain salts at the terminal filaments of the nerves, causing muscular contraction by virtue of the difference created in surface tensions, the same or similar influence in modifying the surface tension between epithelial cells and bacterium in the tonsillar crypts may be expected to tell for the penetration or exclusion of bacteria from the tissues. The excitation carried thither over nerve routes, in combination with the excitation of the sympathetic nerve system elsewhere, may easily be imagined due to peripheral or internal irritation in the metabolism of the human body referred to above.



Plate 1—Showing tubercle bacilli passing through the epithelium of a tuberculous larynx.





Plate II.—Carmin granules penetrating and bacteria remaining on surface of tonsils.

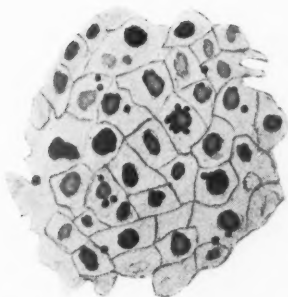


Fig. 1. Showing distribution of fat droplets in an osmic acid section of the faucial tonsil. Oil immersion 1/12.

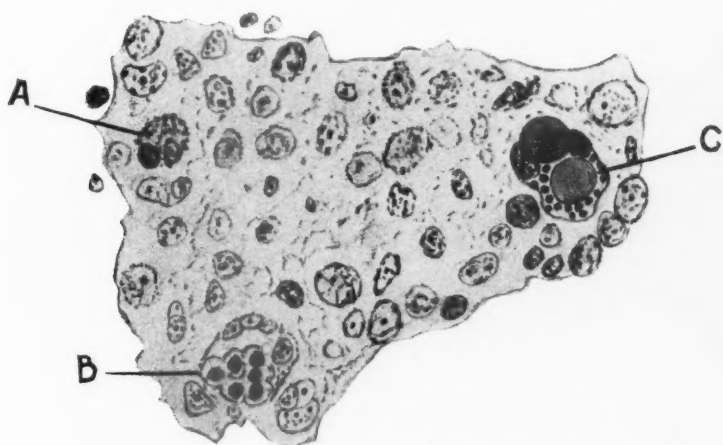


Fig. 2. Showing at *A* a binuclear stipple cell, such as is seen either with the process for staining neutral fat or lecithin with scharlach roth. Oil immersion 1/12.



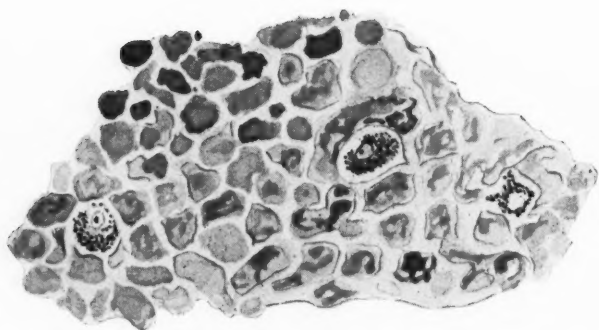


Fig. 3. Showing the mononuclear stipple cells or granulocytes, as revealed by the stains for soap and for cholesterolin.

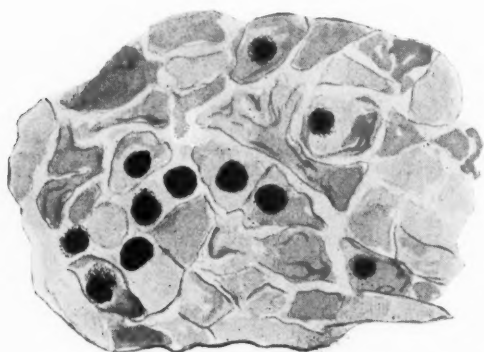


Fig. 4. Showing the round globules of cholesterylolate free in the tissue as a cluster, evidently scattered from some "lipoid cell" in which they originated.

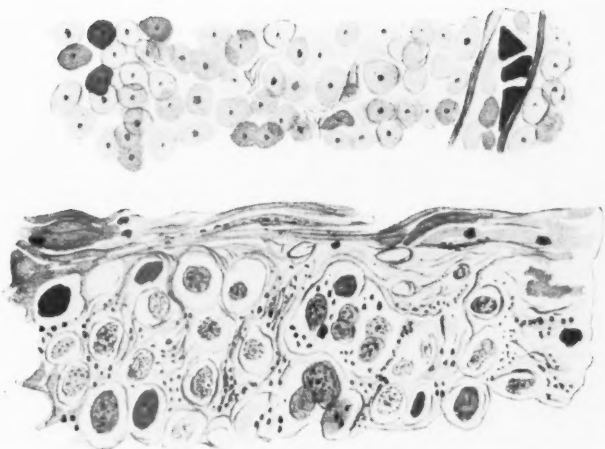


Fig. 5. Showing the distribution of cholesterol in the epithelial layers, and its absence from the stroma just beneath the epithelial area, except for the deep stain of some of the compressed erythrocytes in a capillary.

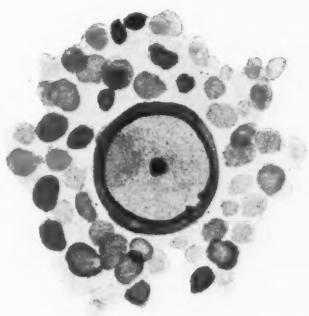


Fig. 6. Showing with the soap stain a ring body free in the tissue of a tonsil which has been removed from the throat under ether.



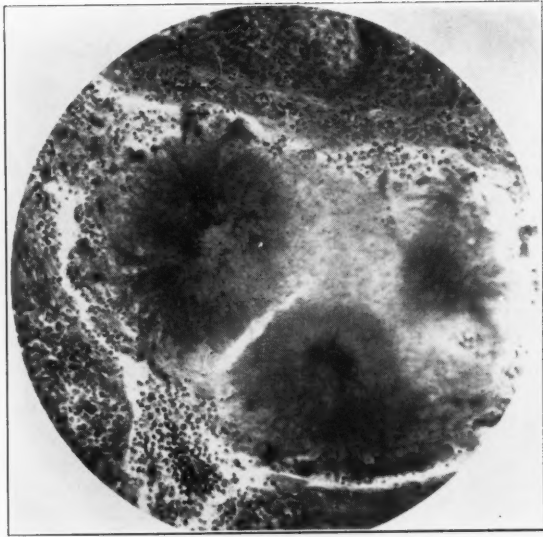


Fig. 7. Photomicrograph of pseudo-actinomyces in tonsillar crypt.
(Lecount.)

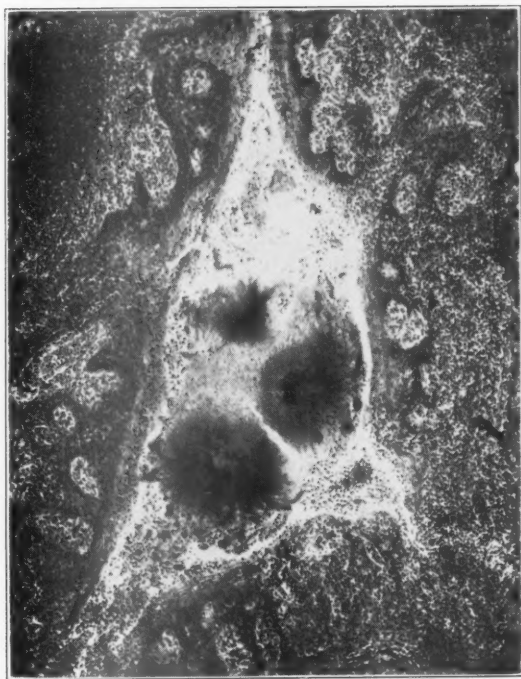


Fig. 8. Photomicrograph of a pseudo-actinomyces in tonsillar crypt.
(Lecount.)



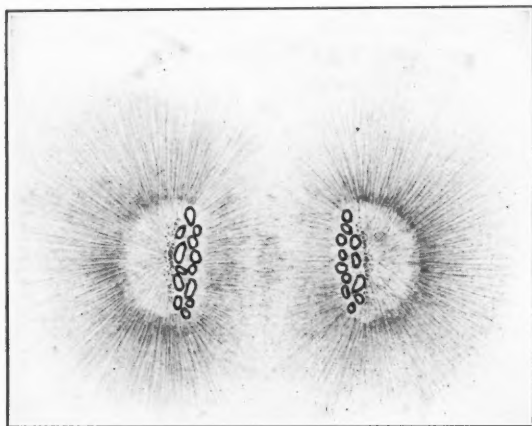


Fig. 9. From Wilson's Cell Division.

VI.

DEGENERATIVE CHANGES IN THE LINING MEMBRANE OF THE MAXILLARY SINUS AND THEIR RELATION TO SYSTEMIC INFECTION.

BY FRANCIS P. EMERSON, M. D.,

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Pathology.—On inspection after an opening through the canine fossa the mucoperiosteal lining may look smooth, may be covered with small, multiple abscesses, a polypoid mass be found in the floor of the antrum or opposite the middle turbinal body and the cavity may be filled with serum or contain a gelatinous mass. The whole mucosa is undergoing degenerative changes, is thin and shows chronic infection throughout. Pus may be found between the mucoperiosteum and the bony wall or an osteitis be present. There may be a thickening of the wall of the blood vessels entering the bone. Sometimes the whole cavity is filled with mucus cysts. While most cysts contain clear serum, occasionally one will contain mucopus tinged with blood. Polypi may be of the mucoid type or contain a large amount of fibrous tissue or become calcified. These are the findings and pathology of the terminal stage. In many cases only a smooth lining membrane is found, which, to the inexperienced, may look almost normal. It is slightly pink in color, and on elevation it is found to be loosened in the bowl of the antrum. On section, chronic inflammation is found to be present. Between these extremes all degrees of atrophy are present, but the writer has always found the process diffused and not circumscribed in character.

Diagnosis.—A careful history going back to childhood should be taken of any general disease that the patient may have had without calling attention to any local trouble. This will disclose, if the antrum is giving trouble, that the patient has suffered at intervals from what seemed to be a cold in the head that was not very marked, but each time it was followed by a myositis, arthritis, asthma, endocarditis, or some gen-

eral trouble that might be the result of some localized infection. In many cases the patient will say that he used to have a thick postnasal discharge, especially in the morning, but of late years it is watery and does not amount to much.

Local Examination.—The nasal mucosa is more injected on the involved side, is often dry or a thin, mucoid coating may be found on the lower part of the inferior turbinate. There is often an associated ethmoiditis with polypi. The turbinal bodies are diminished in size, and the mucosa is the seat of a circumscribed or diffuse atrophic process. The ostium is enlarged and easily entered. On washing out the antrum through the ostium a gelatinous mass is found or only a serous fluid is present or the lavage may be negative. Transillumination is more clear on the affected side on account of the atrophy. The X-ray findings may show polypi or an osteitis, or may be misleading by showing what seems to be a slightly thickened membrane on the good side. There is usually an enlargement of the tonsillar gland on the infected side and a lateral pharyngitis.

Symptoms.—Many patients are free from symptoms until they have an acute exacerbation, which they consider a cold, and to which they are susceptible. The discharge, being serous, is not noticed, and the symptoms are those of a subacute nasopharyngitis. The recurrence of these symptoms may continue for years, or, in exceptional cases, the acute phase may be accompanied by albumin in the urine and symptoms of general toxemia, as in the cases with a thickened membrane. In others the symptoms are those of the systemic complication. Usually, there is no pain over the affected antrum, but is rather constant over or through the eyeball or in the temporal region. Many have vague feelings through the face which are aggravated by changes of temperature, fatigue or any mucous membrane irritation. These feelings of tightness in the face or the localized pain often develop into a trifacial neuralgia. From the involvement of the mucous membrane there may be a subacute conjunctivitis or, through the circulation, a uveitis or systemic manifestation in some distant organ or in special muscle groups. Any change in the circulation of the blood or lymph at the base of the skull is followed by reflex symptoms. Stasis, pressure or any condition which would cause hyperemia

often is the cause of vasomotor symptoms. Fatigue is pronounced. The lymphatic circulation, as worked out by Mullin, would lead us to expect involvement of the bronchial and mediastinal glands, and in the cases with chronic bronchitis, bronchiectasis, in such long standing conditions, and such cases are seen especially in adults with asthma. On the other hand, there are many cases that have extended over years without these changes becoming a factor of sufficient importance to prevent their symptomatic recovery if the upper respiratory tract is relieved of its chronic infection. One class of cases are simply inconvenienced from what seem to be frequent colds. A second class suffer from headaches, neuralgia, fatigue or a bad feeling head; while a third class are invalids from a blood stream infection of some distant organ.

Treatment.—Many still consider these cases as representing the last stage of a run out infection, and even if they occasionally flare up that they do no particular harm. Clinically, they are all dangerous because they represent various depths in the involvement of the lining membrane by chronic infection, and the tissue changes have resulted in damage beyond the hope of repair. Any lining membrane that is left is always a menace to the patient, particularly if there is already communication with the blood stream and the patient is suffering from a general systemic disease. In empyema it is advisable to use every means at our command to cure our patient with the least damage possible to the tissues, in the hope that function may be restored without resorting to a radical operation. In degenerative cases the function is already lost.

Much has been written about infections in the teeth and lymphoid tissue and their relation to general diseases. In chronic infection of the maxillary sinus it was thought by many that a window resection of the antranasal wall, that provided ventilation and drainage, was all that was necessary to render them symptom free, especially in cases with no free pus. There are those who still feel that no chronic case ever gets entirely well, while others believe they can do all that is necessary through the nose when operative measures are indicated. Many cases are irrigated for years, although washing out the antrum never cures a chronic case. For ten years the writer has elected to do the Caldwell-Luc operation.

In this type, where the lining membrane has undergone degenerative changes, the mucoperiosteum is diffusely involved, and when we have systemic symptoms following acute exacerbations of the local chronic inflammatory process, the entire removal of the lining membrane is the only treatment. This should be an interval operation, not earlier than four weeks after an acute phase. On account of the involvement of the deep tissues it is evident that simple ventilation and drainage must be inadequate after a blood stream infection has occurred.

Operation.—The Caldwell-Luc operation is well known, and only certain details need to be discussed at this time. In making the initial incision over the canine fossa, do not come toward the median line farther than the second bicuspid tooth. This will avoid the anterior dental nerve supply and prevent numbness in the teeth and lip. The canine fossa opening should be large enough to allow inspection of the entire cavity. After drying, begin the elevation of the mucoperiosteal lining at its junction with the bony canine fossa opening below. After elevating the lining membrane from the floor of the cavity, grasp the flap with nasal forceps and sweep a curved elevator from side to side under a little traction. Be sure the anterior wall is denuded. Take down the antranasal wall with a chisel through the canine fossa opening and with Greenwald forceps extend the opening back to the posterior antral wall. See that the membranous opening is as large as the bony opening. Lastly, be sure that the inferior turbinate does not rest on the floor of the nose or is not enlarged anteriorly to prevent free access to the cavity later.

After Treatment.—If the above details are observed, no after treatment will be needed in the usual chronic case. When there has been a long standing degenerative process many of them will need an application on a cotton tipped applicator of 25 to 90 per cent silver two weeks after operating if the patient commences to have symptoms. There is no retention of secretion but the granulating surface is congested. It does no harm to pass a cotton tipped applicator through the cavity occasionally, but if it is doing well the less it is disturbed the better. Washing out increases the surface congestion, and the cavity should be wiped dry before an application is made or the patient will complain of a fresh cold that followed the last

treatment. The worst cases we have should not have any symptoms after the first year, but will need attention at intervals during the first three months and, later, whenever they have a fresh infection, until a firm epithelial covering forms. If they are not doing well there will be a thin mucoid coating along the lower border of the inferior turbinate and a return of their old symptoms or those of a fresh cold. There will not be any free secretion in the nose or antrum. These patients should report at intervals and be instructed to come oftener if there is an increase in the discharge, symptoms of a cold or a return of their head pains after the cavity is dry. When the lining membrane has been entirely removed there is not the same tendency to long continued suppuration and the cavity keeps growing smaller. If, on the other hand, any chronically infected mucoperiosteum is left, any previously existing neuralgia pain will persist, notwithstanding that ventilation and drainage are free. Any pathology in the nasopharynx should be corrected and follow up treatment carried out until the thickening and congestion of the nasal mucosa has been relieved. This can be done with Dowling packs and a spray of menthol, grains 2, to an ounce of albolene. This relieves the blood and lymph circulation and indirectly helps the healing process in the antrum, for the nasal mucosa is essentially the same tissue as the lining membrane of the antrum and is thickened in the same way, extending over a long term of years. In fact, some investigators believe that the process starts in childhood from some unknown etiologic cause. While it is true that many do start in early life, the writer has always found that they were due to infection, although the X-ray frequently fails in confirming the diagnosis. No surgical treatment alone ever cures these cases.

Case 1.—Maxillary sinusitis with polypoid degeneration. Mrs. A. L. R., a widow, aged forty-five years, American, assistant housekeeper in a hotel. Referred by Dr. B., August 28, 1924, for deafness, clicking sounds, tinnitus in the left ear and constant head pains.

Present history: Whooping cough, measles, chickenpox, rheumatism and an operation for appendicitis four years ago. She had an abdominal operation three years ago (hysterectomy) and a tonsil and adenoid operation four years ago.

There was some indefinite ear trouble twenty-one and ten years ago. Two years ago there was similar trouble, which she described as a snapping noise in the head. There was vertex pain. On windy days the head feels bad. The throat is rough and there is pain in the arms and shoulders. There is a tightening of the whole head, especially over the eyes, before a storm. Occasional spots before the eyes, particularly before the tonsils were removed. The muscles are lame. The upper teeth have all ached, but the dentist could not find any trouble. One infection last year was followed by wheezing. There was no history of thick nasal discharge until ten years ago. Does her work, but tires easily and has felt miserable most of the time for ten years. The patient is not of a neurotic temperament. She sleeps poorly.

Examination: Nasal mucosa shows chronic infection on both sides. High deviation of the septum to the left. Transillumination of the sinuses is negative. The glands in the neck are palpable. X-ray of the teeth and sinuses had been made several times and pronounced negative. Temperature 99° F., pulse 96.

X-ray report (another): The ethmoid cells are of equal density and normal except for unequal size. The maxillary sinuses are radiolucent, but there is a distinct thickening visible around the walls, which is especially noticeable on the right side.

Clinical diagnosis: Deviation of the septum to the left. From the evidence of nasal infection, the referred pains and the X-ray report, a diagnosis was made of double maxillary sinusitis. This seemed more probable as the patient, without being neurotic, was miserable most of the time with a negative physical examination and all her symptoms referred to the head. She also had an afternoon temperature, so often found in cases with a chronic infection.

Operation: December 6, 1927. Double Caldwell-Luc operation. Preliminary medication, atropin, grs. 1/150. Gas-ether anesthesia. Postnasal packing inserted. The right antrum opened through the canine fossa, the mucous membrane found to be atrophied and stripped readily from the bone. There were two troublesome bleeders, one from in the nasointral wall and one in the lateral wall.

The nasoantral wall was taken down, the antrum packed with vaselin gauze and the incision closed by two catgut sutures. The left antrum was opened in the usual manner, the membrane removed, and packed with vaselin gauze and the incision closed by two catgut sutures. During the entire operation she bled profusely from the membranes, and the blood did not show any tendency to clot. Two cc. of hemoplastin given during the operation. Postnasal packing removed and the patient returned to bed and a shock stimulation given.

Progress: December 6, 1927, 3:00 p. m., patient was oozing from the nostril, and the anterior nose was firmly packed; 5 cc. of thromboplastin ordered. December 6, 1927, 6:00 p. m., slight oozing from the nose; 5 cc. of thromboplastin given. December 7, 1927, 2:30 a. m., pulse 120, patient vomited bright blood. Examination showed postnasal bleeding. Postnasal packing inserted. Anterior nose repacked. December 7, 1927, 10:00 a. m., patient has had no bleeding during the day. December 8, 1927, packing from the anterior part of the nose removed, postnasal packing removed. Packing from the antrum removed under gas-oxygen anesthesia, followed by considerable bleeding, which, under ice compresses, seemed to be controlled. December 8, 1927, 1:00 p. m., vomited quite a quantity of bright red blood. Examination showed postnasal bleeding; postnasal packing reinserted, and the nose packed anteriorly. Pulse 120, and was quite feeble. Patient exceedingly anemic. Dr. S. requested to type for transfusion and to give transfusion. December 8, 1927, 9:00 p. m., patient type No. 4, 600 cc. of blood given by Kimpton tube method. December 9, 1927, patient's condition greatly improved and she has had no more bleeding. December 10, 1927, the packing was removed from the nose anteriorly; there was bleeding on the left side, which necessitated packing along the floor. December 10, 1927, 4:00 p. m., packing along the floor of the nose removed and the postnasal packing removed with no bleeding. December 12, 1927, patient's pulse has dropped to normal, temperature is normal, has had no bleeding since the removal of her packing, and her condition is excellent.

Blood examination: Differential count of 100 leucocytes, polymorphonuclear neutrophils 72 per cent, lymphocytes 19, large mononuclears 7, eosinophiles 2. No obvious leucocytosis

or leukopenia. Red corpuscles appear normal in smears. Platelets apparently increased in numbers. Bearing in mind the limitation as to what can be inferred from inspection of a smear alone, leukemia and other related conditions can be ruled out. Also thrombopenic purpura hemorrhagica can be ruled out. There appears to be an increase in platelets, a perfectly normal reaction to hemorrhage. With the coagulation time of three minutes, hemophilia can also be excluded. Not much reliance can be placed on the clotting time, as judged from the behavior of blood exuding from a wound and coming through the nose. Also, hemophilia is extremely rare in women. There are, however, a great many cases of hemorrhagic disease which do not run true to form. (Dr. L.)

Case 2.—Antra with thickened lining membrane and beginning degenerative changes. Mrs. J. B. A., aged fifty-four years, housewife, American. Referred by Dr. M., June 24, 1925, for nasal obstruction.

Present history: Bronchitis, asthma, grippe. The patient had scarlet fever twenty-three years ago, grippe several times, and asthma for twenty years. Fatigue was marked.

Examination: Septum to right. Left mucosa injected. Left middle turbinate has been partially removed. Transillumination was dark over the left antrum and the eye reflex was not present. There was more discharge from the left nose and more tenderness over the left canine fossa. No adenopathy present. The tonsils showed chronic infection.

X-ray report: All the sinuses were clear except the antra, in both of which there was a very slight thickening of the membrane. On the left side there was sclerosis of the bone under the lining, suggestive of some previous infection.

Clinical diagnosis: Right chronic antrum with degenerative lining membrane. Left chronic antrum with thickened lining membrane.

Operation: June 30, 1925, at the Brooks Hospital. Preliminary medication, atropin, grs. 1/150. Gas-ether anesthesia. The double radical antrum operation (Caldwell-Luc) was done. Polypoid membrane found in both antra. Vaseline gauze packing and incision closed by catgut sutures. Tonsillectomy. Two sutures required in the left fossa.

Progress: April 15, 1927. Twenty-two months after operation the patient states that she has had but one attack of asthma and has had a better winter than for years. No antral discharge.

Case 3.—Antra showing chronic, inflammatory changes in the lining membrane with secondary atrophy. Miss E. M. M., aged twenty-seven years, American, nurse.

Complaint: Constant colds, specks before the eyes, with repeated attacks of myositis.

Present history: Grippe, bronchitis, pneumonia, appendicitis, rheumatism, measles, mumps, whooping cough, chickenpox, bronchitis. Always had colds and bronchitis nearly every winter. She had occipital headaches as a child and laryngitis since fifteen years of age. The patient had a tonsil and adenoid operation in 1923, and has had no loss of voice since but gets very hoarse. There was an attack of bronchitis in 1923, with an acute appendix, but no operation. In 1920, the patient had lobar pneumonia, and in 1921 bronchopneumonia. Also, an acute antrum in 1921 and 1924, on the left side, with myositis in back and shoulders and a good deal of foot trouble (flat feet). During the last two years there has been constant colds and hoarseness, especially when tired, with repeated attacks of myositis. The patient has been having specks before her eyes, which the ophthalmologist says is due to infection, with headaches four or five times a week.

Examination: The left nasal mucosa was injected. Transillumination too clear on the left side and through the base. There was no tenderness on pressure. There was a palpable gland in the neck and no free secretion in the nasopharynx. The right nares were negative.

X-ray report: The frontal sinuses are small. The other sinuses are perfectly clear. (Macmillan.)

Blood examination: Hemoglobin 87 per cent, red count 4,080,000, white count 8,355. Differential count: Polymorphonuclears 64 per cent, large lymphocytes 28, small lymphocytes 7, mononuclears .5, mast cells .5.

Clinical diagnosis: Left chronic maxillary sinusitis with beginning secondary atrophy.

Operation: The radical Caldwell-Luc operation was done under gas-ether anesthesia, and entire lining membrane re-

moved. Upon removing the bony wall the membrane covering the face of the antrum stripped readily from the bone, ballooning into the antrum. Bone and membrane sent to the laboratory.

Laboratory report: Specimen submitted shows chronic inflammation of the mucosa and periosteum, with some inflammatory reaction in the blood vessels of the bone. There is no reaction in the bone tissue proper. (Slack.)

Case 4.—Mrs. M. F., a widow, aged fifty-three years, American. Referred by Dr. L. for headaches and trifacial neuralgia. Duration, eighteen years. Patient is well nourished and of good color.

Present history: A frontal sinus and radical antrum operation, as well as a tonsil and adenoid operation and a submucous resection of the nasal septum, were done three years ago. An abdominal operation (hysterectomy) was done eight years ago. All the teeth had been removed. The patient suffers from continuous colds. The headaches are frontal, over the left antrum, behind the ear, and when bad there is occipital pain. She is never wholly free from pain in one of the places. Fatigue is marked. The patient sleeps poorly. There is crusting in the left nares and postnasal discharge. Indigestion or myositis are not present. Several Wassermann tests have been negative. The patient is unable to apply herself to her work.

Examination: More sensitive to pressure over the left antrum. Clear pus in the middle meatus on both sides. Posterior nares free and pharynx smooth and clean. There is a large opening in the left middle meatus and under the inferior turbinate through the antranasal wall. The anterior end of the right middle turbinate is wedged in between the septum and the outer wall. There is probably infected membrane on the anterior wall of the left antrum, and some of the ethmoid cells on the same side have not been cleared out. The right antrum is in doubt. The nasal mucosa is much thickened and shows chronic infection. A cotton wipe passed over the outer face of the sphenoid, along the lower border of the inferior turbinate or over the bowl of the left antrum, starts up nerve pain that is acute.

Clinical diagnosis: Trifacial neuralgia. Residual infection in the left antrum and nasal mucosa. Some ethmoid cells not cleared out.

Operation: March 9, 1927. Double Caldwell-Luc operation. Preliminary medication, atropin, grs. 1/150. Gas-ether anesthesia. Postnasal packing inserted. The left antrum opened through the canine fossa. The mucous membrane elevated, and an old opening found in the antrum made at a previous operation. This opening was enlarged and the antrum explored. Most of the pathology in the left side was found in a big depression over the molar teeth. The opening into the nose was enlarged anteriorly and taken down to the floor of the nose. Granulation tissue over the left posterior ethmoid cells, which was removed. The right antrum opened and the mucous membrane found to be normal. Both antra packed with vaselin gauze, the incisions closed with two catgut sutures. Postnasal packings removed. Pressure bandage applied.

Progress: April 4, 1927. Is having boils under the arm. April 20, 1927, pain decidedly worse following the operation. Especially pronounced in the head and back of the ear and in the throat. May 7, 1927, is sleeping better. June 7, 1927, anterior end of right middle turbinate removed, and it was found adherent to the septum. July 7, 1927, the nasopharynx has been treated weekly and applications of 25 per cent AgNO_3 made to the bowl of the left antrum. In the left sphenoid the upper two-thirds appears normal, but the lining membrane below is distinctly of a sleazy appearance. The anterior wall is removed and trichloroacetic acid applications made to the lining membrane. The area over the anterior wall is very sensitive, even after an application of 10 per cent solution of cocaine. July 21, 1927, the surface infection has disappeared everywhere except at the lower part of the sphenoid. November 2, 1927, is sleeping well and has no pain except at long intervals. December 15, 1927, no pain. Discharged.

Case 5.—Double chronic maxillary sinusitis with secondary atrophy on the right side. Miss E. B., aged thirty-eight years, a teacher (American), was first seen on January 3, 1927, with the question of some focus of infection.

Present history: Tonsillitis followed by repeated attacks of myositis. No colds until last winter. Influenza, 1919 (mild

case). In 1923 she commenced to have trouble in voiding. No sore throat preceded it, but there was a bad taste. Six years ago she had indigestion and has to be careful now about her diet. There was occasional acidity. She has had myositis in her right shoulder and is having a little stiffness of the neck now. She has severe generalized headaches, accompanied by pain in the teeth on the right side. Her tongue gets coated. She has a bad taste and her stomach is upset as often as once a month for the past fifteen years. Her face is sore with her colds. She has backache every night. She was examined by two rhinologists, who pronounced the nasopharynx and sinuses negative.

Report of general examination: Two years ago she complained of frequency of urination and bladder pain. Examination locally showed ulcer in the apex of the bladder. Local treatment for a year was followed by no improvement. On June 14, 1926, a large portion of the apex of the bladder containing two ulcers was resected. There was some improvement in the pain and frequency of voiding, but the urine continued to show infection, and the results were only partially satisfactory. Examination on November 20, 1926, showed definite strictures in the lower end of both urethers and pus coming from both kidneys. The ulcers had healed and the bladder showed no other trouble. During these sixteen months she had been referred to two rhinologists and a dentist, who failed to find any source of infection, although the urologist felt that this class of case always results from some primary focus.

Local examination: Her left neck showed a palpable tonsillar gland and both tonsils showed chronic infection. The right antrum is more clear to transillumination, especially through the base. The nasal mucosa was negative. There was no pain on pressure over the canine fossæ. There was a right postnasal spur. There was postnasal secretion and none in the nares.

X-ray report: The left mucoperiosteum definitely thickened in the maxillary sinus (Macmillan).

Urine examination. January 7, 1927. Reaction was slightly acid, with specific gravity of 1006. There was no sugar or acetone. There was a slight trace of albumin (Heat — HNO_3)

and a small amount of pus but no crystals or casts. There were many squamous cells and, rarely, a large round epithelial cell.

Diagnosis: From the clinical history, confirmed by the finding of a palpable tonsillar gland on the left side, and local evidence of chronic infection, together with evidence of abnormal clearness to light through the right antrum, where she had the pain in her teeth, and the evidence from the X-ray of a thickened membrane, a diagnosis was made of chronic tonsillitis and double chronic maxillary sinusitis with probable secondary atrophy on the right side.

Operation: January 7, 1927. Tonsillectomy and double radical antrum through the canine fossæ, under general anesthesia. Entire lining membrane was removed and this, with a section of bone, was sent to the laboratory. Mucoperiosteum was thicker on the left side, and both sides showed chronic infection of the deep tissues without involving the bone. (Slide.) (Slack.)

Postoperative result: February 17, 1927. Urine examination the same, except an increase in specific gravity to 1010 on three examinations. Backache was relieved at once and the patient has had but one headache. Pain on voiding was gone but frequency remains the same.

Case 6.—G. A. M., aged forty years, single, Irish American, clerk in freight office. Referred by Dr. L., November 30, 1927.

Complaint.—Unable to use his eyes for a year on account of a subacute conjunctivitis. Head pains.

Present history: Has a cold with every nasal irritation, whether from dust, heat or exposure. Left nose obstructed. Has what the patient describes as a funny feeling in his face, which starts in the forehead and passes over the entire face. When this is bad the pain extends to the occiput. A hot room affects him badly. The eyes and feeling in his face are worse on lying down. This bad feeling is sometimes accompanied by tinnitus in the right ear and vertigo, and becomes bad enough at night to make him sit up to get relief. A diagnosis of right maxillary sinusitis was made two months ago, and the antrum was opened under gas and washed out repeatedly. The discharge has been less since, but not the bad feelings.

Nine teeth have been removed at different times. Previous tonsil and adenoid operation.

Examination: Septum to the right. Both antra dark to transillumination. Mucosa slightly injected in both nares. Right tonsillar gland used to swell before his teeth were extracted. Is now palpable. X-ray shows a thickened lining membrane on both sides.

Clinical diagnosis: Double maxillary sinusitis with secondary degeneration.

Operation: December 8, 1927. Double Caldwell-Luc operation. Preliminary medication, atropin, grs. 1/150. Gas-ether anesthesia. Postnasal packing inserted. Membrane found to be diseased in both sides. The membrane was removed and sent to the laboratory for examination. Vaseline gauze packing and incision closed by catgut sutures. The postnasal packing removed.

Progress: December 10, 1927. Patient is doing satisfactorily and there is very little discharge from the nose. December 19, 1927, the eyes are better. There is only occasional head or face pain. There is some discoloration beneath the right eye.

Case 7.—Miss P. S., aged thirty-two years, American, executive secretary, referred by Dr. L.

Complaint: Spots before the eyes and headaches over the eyes.

Past history: Children's diseases, grippe, bronchitis, tonsil and adenoid operation in 1924, both otitis media acute at fourteen years of age, and colitis one summer.

Present history: Last June, Miss S. commenced to have severe headaches over the eyes and in the back of the head. More marked in the morning. The patient has had a mild hay fever each summer. No colds since the tonsil and adenoid operation. There was a history of a bad bronchial cough two years ago. Two months ago both antra were washed out to see if it would help her headaches, although she was not having any antral symptoms.

Examination: Right antrum transilluminates too clearly. On the left side the eye reflex does not show. The right nasal mucosa is injected and the inferior turbinate is enlarged. The septum is deviated to the left. Transillumination through the right base is too clear and the light passes out into the right

alveolar process. The examination of the blood is negative. There is no lymphocytosis. (Polymorphonuclears 57 per cent, small lymphocytes 27.5, large lymphocytes 9.5, transitional 3, eosinophiles 3).

X-ray report: No sinus trouble (Macmillan).

Operation: January 27, 1927. Double Caldwell-Luc operation. Preliminary medication, atropin, grs. 1/150. Gas-ether anesthesia. Postnasal packing inserted. Usual incision made through the right canine fossa. Specimen of bone removed for pathologic examination. The lining membrane appeared healthy. On touching it with a probe it appeared to be normal in character. The nasoantral wall taken down, cavity packed with vaselin gauze. Incision closed with two catgut sutures. The left antrum showed a chronic atrophic membrane. It stripped from the floor and from along the nasoantral wall. Specimen was removed for pathologic examination. Cavity filled with vaselin gauze packing. Incision closed with two catgut sutures. Postnasal packing removed.

Laboratory report: Specimen from both antra shows chronic inflammatory reaction of the mucosa and periosteum. There is noticeable increase of the fibrous tissue in the specimen from the left side. There is no evidence of inflammatory invasion of the bone in either specimen. (Slack.)

Progress: February 17, 1927, sinuses clean. No pain, eyes better. March 24, 1927, eyes keep improving. No discharge. October 3, 1927, eyes much better. Discharged relieved.

VII.

THE LOCATION OF THE FOCUS IN OPTIC NERVE DISTURBANCES FROM INFECTION.*

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While meditating on a title for this paper in which I planned to evaluate the various conceptions of etiology of optic neuritis, I turned for inspiration to one of my old standbys,—“Focal Infection,”¹ by Billings, and ran across the following: “When a systemic disease occurs which present day knowledge associates with a primary infectious focus, the site of the focus must be located.” If it is of primary importance in general medicine to determine the site of the infection before successfully treating systemic disease, is it is not equally important for rhinologists to find the focus whenever treating blindness or deafness which present day knowledge is beginning to attribute to a primary infectious focus? The sinuses, because of their propinquity to the optic nerve, have from the first absorbed the attention of both ophthalmologists and rhinologists. Early writers and even many of today direct their attention almost exclusively to the adjacent sinuses. The other foci so frequently found of major importance in general medicine are neglected. I believe that it is well worth while to consider these various foci of infection.

Whenever a patient with a neuritis presents himself to an expert diagnostician there are certain lines of investigation that are almost universally followed. The teeth, according to most investigators, among whom Rosenau occupies a conspicuous place, have been found most frequently to harbor the infecting focus. Tonsils are rather a close second, while the sinuses are a poor third, possibly not even holding that position. The appendix, prostate, fallopian tubes, gall bladder, etc.,

*Read before the Laryngological Section of the New York Academy of Medicine on November 23, 1927.

must all be considered. A primary focus may have produced so many secondary foci that its elimination does not cure the neuritis. The infection is believed at present to usually travel by way of the blood stream.

In considering the history of these optic nerve disturbances we go back about one hundred years when no one dreamed of the possibility of systemic infection ever arising from such an insignificant thing as a tooth. The sinuses, because of their propinquity to the optic nerve, naturally offered the best solution of the etiology of sudden loss of vision. The relief frequently following any one of the various surgical procedures on the sinuses seemed to prove this theory.

While there has been a steady accumulation of data on the relationship between eye diseases and infections, it is but recently that anything like a scientific explanation could be made. It was unfortunate in a way that the sinuses should have been the first to receive attention, although we owe much to the brilliant and painstaking investigations of Onodi,² who in 1908 published his epoch making study on the "Anatomic Relationship Between the Accessory Sinuses and the Optic Nerve." Later contributions by Loeb³ and many others greatly added to our comprehension of this subject. The sinuses, therefore, largely from this priority of investigation, occupied for a long time the supreme position in the minds of most rhinologists. Until recently we were unable to exclude them as the principal focus of infection. It was not until radiography had reached its present stage of development that infections within the various sinuses could, with any degree of accuracy, be located or excluded.

I frankly admit that when I commenced to study these cases, and for some years thereafter, I expected to find the explanation for all optic nerve disturbances within the sinuses. The literature at that time, as far as I can recall, dwelt exclusively on the importance of the sinuses. I was told by the ophthalmologist who referred my first case that there would be permanent loss of vision if I did not open the sphenoid within twenty-four hours. His advice was followed and I was gratified with the rapidity with which the vision was restored. What better proof than that the cause of the infection was in

the sphenoid, and that in opening it the focus had been eliminated? The sphenoid did not appear diseased. It was lined with a thin white membrane and contained no secretion. Other cases followed, most of which made speedy recovery. The sphenoids rarely showed anything abnormal. In order to put this work on a more scientific basis I saved the tissue for microscopic study and made cultures from the sinuses. While occasionally some changes from the normal were discovered, this tissue did not differ especially from what one would find on examining similar tissue from an equal number of individuals without visual disturbances. The radiographs of these sinuses were also disappointing. Dr. A. S. Macmillan, the radiologist at the infirmary, summarized the findings as follows:

"The radiographic examination in this group of cases was rather disappointing from the standpoint of one seeking definite pathology. In a number of instances, no clouding could be made out in the sphenoid or ethmoid cells; in others, a slight blurring of the cell partition was evident, while in none of the cases was there definite demonstrable pathology."

This influenced me to look for other sources of infection. The benefits following the removal of infected tonsils in one or two otherwise negative cases led me to consider them as a possibility in every case. Another case with self evident dental infection directed my attention to the teeth, while a patient with some pelvic infection, who developed a retrobulbar neuritis and was relieved by the removal of the tubes, led to the consideration of the more remote regions of the body.

Gradle,⁴ in 1915, was among the first to revolt against the prevailing theory that these optic nerve lesions resulted from the close relationship between the sinuses and the optic nerve. He said: "I believe that I am justified in stating" (contrary to the teachings of Onodi and Loeb) "that the anatomic relations of the sphenoid and ethmoid cells to the optic canal are immaterial when it comes to a question of optic nerve involvement in accessory sinus disease."

When these vision cases were relieved by the removal of tonsils or teeth, the prevailing theory that there was a direct extension from the infection in the sinuses to the optic nerve needed amending, and, as blood stream infection by that time was well recognized, this was adopted as a possibility. The

hematogenous route is today considered the almost exclusive method of infection. In a former paper,³ presented before the American Laryngological, Rhinological and Otological Society in 1921, I quoted the following from Billings, and as I still believe it is apropos, I repeat it:

"Systemic infection and intoxication from a primary focus is usually hematogenous. The bacteria may be compared with emboli loosened from the place of origin and carried in the blood stream to the smallest and often terminal blood vessels. If virulent and endowed with specific elective pathogenic affinity for the tissues in which they will lodge, and if in sufficient number, the invading bacteria will excite characteristic reactions in the infected tissues and a sequential train of morbid anatomic lesions. The evolution of the anatomic lesions and the clinical phenomena aroused thereby are dependent on the type and virulence of the bacteria, the character of the tissue and the function of the organ involved. The specific tissue reaction consists of a local inflammation with endothelial proliferation of the lining of the blood vessel, with or without thrombosis; blocking of the blood vessels; hemorrhage into the immediate tissue; positive chemotaxis with resulting multiplication of the leukocytes and plasma cells in the infected area, or fibrinoplastic exudate with local connective tissue overgrowth. Hematogenous focal infection of the nervous apparatus, involving the gasserian and posterior spinal root ganglia and spinal cord, affords confirmation of the infectious nature of herpes, of insular sclerosis and myelitis of the spinal cord. Removal of the primary etiologic foci of infection about the upper air tract and mouth may modify favorably the course of the spinal cord infection, and if spinal cord infections, why not, let me add, similar infections of the optic nerve?"

You physicians probably know vastly more about hematogenous infection than I do, so pardon me for dwelling upon it. It is so well recognized today that it needs no argument that I could present to substantiate it. The point I wish to bring out is that practically all types of neuritis, no matter whether they are of the ophthalmic, the trifacial, the auditory or the ulnar nerves, arise from some focus of infection. It would be a good thing for our comprehension of the etiology of these optic nerve disturbances if the sinuses were removed

to the most distant part of the body. Then they could be evaluated from the medical man's point of view for the explanation of a neuritis. I have never seen a case, except in malignant disease of the sphenoid, where I thought there was direct extension from the sinuses to the optic nerve. I believe it to be always hematogenous. This has been my belief for the last two or three years, and the cases coming to me have been handled accordingly. The sinuses have been considered only as one of several possible foci and then only as a minor possibility. I have discarded my efforts to find in them some mysterious infection that does not show on a radiographic plate. If transillumination and radiographs confirm my findings on inspecting the nasal cavities and these are negative, I immediately look elsewhere for a focus.

Of late the teeth have furnished the foci in so many cases that it is really a great disappointment when a patient comes who has lost all his teeth or with teeth so healthy that there is little hope of finding the trouble there. Because of the frequency of dental infection and the ease with which it is overlooked and also because little or nothing can be added to your knowledge of the foci in the tonsils or sinuses, I wish to discuss dental infection at some length. I was formerly satisfied to eliminate the teeth from further consideration when patients informed me that their dentists had pronounced their teeth perfectly all right. I happened to have about this time some personal experience with dental infection. My knees became swollen and painful. My teeth had always received attention semiannually and appeared perfectly healthy. None of these teeth had troubled me for some years, but as my tonsils and sinuses appeared negative I had the teeth filmed, and when extraction of two was advised because of apical infection I could hardly credit the report. The removal of these teeth relieved the knees. From then on I had the teeth filmed in all my optic nerve cases, and when these were negative for apical infection or pyorrhea the teeth were dismissed. It was not until I had a recurrence of the knee infection that I learned something more about teeth. In this latter instance the dental films were negative, yet Dr. L. M. S. Miner, who examined both teeth and films, felt that two of the teeth were clinically unhealthy and their removal was followed by prompt

relief. Two years later there was a recurrence, which was relieved by the extraction of three teeth, negative as far as filming went. This has taught me from personal experience, which is my best teacher, that it is no sinecure to determine whether or not a tooth is at the bottom of one's trouble. Apical abscesses are only one of several factors in dental infection. The pulp and the dentine both harbor microorganisms not always evident on a radiographic film, yet capable of producing most serious systemic infection. I hope to have time to cite cases where teeth were apparently ruled out, yet further investigation by one especially versed in dental pathology revealed the trouble, and removal of this focus was followed by prompt recovery.

The point I wish to stress is that dental films do not tell the whole story. The ordinary dentist is little versed in dental pathology. He devotes his efforts to save teeth. Extraction, in his mind, is only justified as a last resort. Only the man who devotes his time and thought to dental pathology is competent to pass on the health or disease of the teeth. I never feel that the teeth have been properly ruled out until such an expert has reported that they are all alive and free from infection. It is not always pleasant, I assure you, to antagonize the patient's trusted dentist, but the results justify it.

Just a word on devitalized teeth, as it is a subject that creates much discussion among our confreres of the dental profession. Some go so far as to advocate the removal of all devitalized teeth, while others try to differentiate between healthy and infectious teeth. Were I confronted with the solution of such a problem in a patient with serious optic nerve disturbance, where after thorough investigation nothing but devitalized teeth had been found, I should unhesitatingly advocate their removal. Rice,⁶ in a paper on "Focal Infection from the Dental Standpoint," in 1926, aptly states:

"It is significant in the extreme to note that thousands of teeth that are symptom free, and that are roentgenographically negative, are being examined in the bacteriologic laboratory after extraction and found to be infected. In view of this, we are eminently justified in viewing all pulpless teeth with grave suspicion.

"The conservative dentist will say, 'This patient is in good health, the pulpless teeth are symptom free, and roentgenograms are negative. Why, then, sacrifice the teeth?' On the face of it, the reasoning seems sound, but on second thought, shall we retain these teeth until a myocardial disturbance puts in an appearance, or a cholecystitis, a rheumatoid arthritis, an iritis, a corneal ulcer, renal calculi, and so on, or shall we practice prophylaxis and remove the suspicious teeth?

"It is not possible to determine whether a pulpless tooth in the mouth is or is not sterile. Men of high professional standing have wrestled with the problem, but the result of their splendid effort has been far from satisfactory, since the proof of the work can only be accomplished after extraction.

"It is known, beyond the peradventure of doubt, that dental foci of infection are among the most active agents that militate against bodily well being, and graveyards are filled with their victims.

"The roentgenogram is an indispensable adjunct in diagnosis, but its limitations must be carefully considered.

"Prophylaxis is the highest type of medical service, and a courageous application of the principles it involves holds out to the laity an assurance of longer life and greater comfort."

When the teeth, after a real investigation, have been pronounced negative, the tonsils should be investigated as the next most probable focus of infection. It is not always possible preoperatively to determine whether or not the tonsils harbor infection. Special attention is given to any enlargement of the glands below the angle of the jaw. Congestion or swelling about the soft palate or pillars usually indicates infection. The tonsil is probed and pulled into the throat, its size and general appearance noted and suction used to remove any secretion that may be in the crypts. Some tonsils are so evidently diseased that their removal would be indicated, irrespective of the eye condition. In such instances I should advocate their removal, together with any infected teeth. I usually take out the tonsils first and then the dentist extracts the teeth while the patient is under the anesthetic. There are tonsils which are suspicious, but one is in doubt as to whether or not they are really the principal focus of infection. If in such patients some

other definite focus has been found, this other focus is first removed. If the case does not progress satisfactorily the tonsils are removed later. As a matter of fact, I have rarely if ever had to remove them later. With other findings negative, tonsils at all suspicious should be removed. I might add that most tonsils are suspicious until placed in a bottle.

Of the sinuses the antrum is the most common seat of infection. This may only show a thickening of the lining, but if this is marked it indicates definite pathology, even without retained secretion. It would be advisable to give such an antrum ventilation by making a good sized opening beneath the inferior turbinate. More marked antral pathology would call for more radical operative procedures. Any of the other sinuses harboring definite infection should be drained and ventilated. I have made it a rule in these sinus infections to do only what would be necessary had the case not been complicated by the eye condition, but it is essential to do this at once. I have had to open the frontal but once in these optic nerve cases, but should it be chronic and the only focus, I should not hesitate to do so in any case. If the frontal were acute, as happened in one instance, I should attempt to obtain drainage by intranasal procedures. I did this successfully in this one case. Ethmoid infections, at least in my cases, have been rare, and the removal of the middle turbinate when obstructive has generally relieved the situation. Rarely have I found an infected sphenoid, but when present the front wall has been taken down. This is a procedure that I formerly advocated in nearly all cases, but which later has been found unnecessary except in rare instances, as the summary of the last thirty cases will indicate. It may still be applicable as a therapeutic measure in the most severe cases—cases where optic atrophy is to be feared, cases with abnormally small optic canals or cases with complete loss of vision.

Just a word in reply to a question that is always asked in discussing this subject. Why does opening the sinuses seem to benefit so many of these cases? An explanation of this mystery was attempted in a paper⁷ I presented before the American Laryngological Association in Montreal, in June, 1926, entitled "The Influence of Negative Pressure in the Sphenoid on the Optic Nerve." In this article it was explained that the

benefits usually following the opening of the posterior sinuses resulted from the relief of the negative pressure and not from the removal of a focus of infection. It is probable that in some cases the drainage and ventilation of the posterior sinuses is so impaired that a condition of negative pressure results. This would probably favor the migration of bacteria and toxins from the blood stream to the optic nerve, a condition similar to what usually exists in the tympanum in Bell's palsy. Let us also add that the apparent benefits are largely due to the fact that the accessory sinuses are opened at about the time the case would usually commence to improve, and to the depletion of the structures adjacent to the nerve.

In dealing with patients presenting multiple foci, rare good judgment is needed. I have read reports of double ethmoid and sphenoid exenterations combined with the removal of the tonsils. To my mind, a needless and unwarranted procedure. I have never seen a case that justified even a unilateral ethmoid and sphenoid exenteration. In all instances, if teeth or tonsils are suspicious enough to warrant removal, let that be the first procedure. If this does not afford the expected relief, the posterior sinuses may later be opened. In only one or two cases have I combined the removal of teeth or tonsils with the opening of the posterior sinuses, and even then I do not think I gained anything. In a facetious way, in one of my last papers,⁸ I made the following observation:

"The papers constantly appearing on this subject, I regret to say, nearly all advocate too radical intranasal surgery. While some writers merely recommend a unilateral ethmoid and sphenoid exenteration, others are only satisfied with a bilateral one, to which in some instances is added a tonsillectomy. I have been expecting that, in addition to the bilateral exenteration of the sinuses and the removal of the tonsils, someone would advocate extraction of all the teeth. This would seem about as sane as the wholesale exenteration of uninfected sinuses. Teeth that often excel in beauty and utility might replace those extracted, but as yet nothing has been devised to replace the sinuses."

With the remote foci of infection throughout the body I have had but a limited experience. I have referred to the case

where the only focus that could be found was in the pelvis and prompt relief followed its elimination. Postinfluenzal retrobulbar neuritis is, I believe, fairly common, but the ophthalmologists usually look after these cases or refer them to a medical man. Among the last sixty-four cases of my series there were five where no definite foci were found. One was attributed to diabetes, another to hyperthyroidism, the third to scarlatina, the fourth to influenza and the fifth to an acute coryza.

If we concede that optic neuritis may arise from haematogenous infection, it logically follows that it may arise from any condition in the body that is capable of furnishing bacteria or their toxins to the blood stream. At the Dallas meeting of the American Medical Association, in 1926, several extremely interesting papers on iritis were read before the Ophthalmological Section. These are so applicable to the subject under discussion that I cannot refrain from again referring to them, more especially as the percentages of the various foci in iritis are almost identical to those in my last sixty cases of optic nerve disturbances:

A paper⁹ from Iron and Brown, entitled "Recurrence of Iritis as Influenced by the Removal of Infections," deals with fifty cases of iritis, followed from three to twelve years. The probable source of the infection was found in forty-six of these cases. The teeth alone were considered the focus in 20 per cent; the tonsils in 34 per cent; the sinuses (antrum) in 2 per cent; the teeth and tonsils combined in 10 per cent; the sinuses, teeth and tonsils in 4 per cent; and the sinuses, teeth and colitis in 2 per cent. Thus it would seem that sinuses play as minor a rôle in iritis as they do in optic neuritis.

Zentmayer,¹⁰ in a paper entitled "The Prostate as a Remote Focus of Infection in Ocular Inflammations," directed attention to the importance of searching further than the mouth, nose and throat for the portal of infection. He states that it is not infrequently found in the prostate gland.

A third paper by Mills,¹¹ entitled "Ocular Disease Occurring in the Course of Nondysenteric Amebiasis," calls attention to the importance of investigating the intestinal tract. He states that 'it is certain that the arrest or cure of chronic iridocyclitis, choroiditis, retinitis, episcleritis, keratitis and conjunctivitis

has followed the faithful use of ipecac and its derivatives when the recognition of a protozoan or flagellate background has ended repeated vain searches for a bacterial etiology.' He furthermore states that in his sole case of double optic atrophy three varieties of endamebas were found at the first examination of the stools, 'and that all other tests, including roentgenograms of the brain, sella and sinuses, were without results.'"

It may interest you to know that Dr. George Derby's treatment for retrobulbar neuritis, when no definite focus is found, is to put the patient in bed, sweat him and flush out the kidneys and intestinal tract.

By exercising due care in our own examination of a case, by having an expert radiologist pass on the sinuses; and by having competent men go over the other regions of the body, it is practically always possible to determine the source of the infection. The important thing, in treating each and every case, is to eliminate the focus, unless the patient is already much below par. Should he be convalescing from influenza or have some other debilitating disease, there might be danger of losing his life. Three or four of my patients were such poor risks that it was necessary to wait some weeks before attempting anything surgical. Unless some definite focus is found it is best to treat the case rather than to exenterate the sinuses on a mere chance that they might harbor infection. The intestinal tract should be kept healthy and the patient put in the best possible condition to combat his infection, as in many instances retrobulbar neuritis only comes when resistance is low.

In cases where I suspect impairment of the drainage and ventilation of the posterior sinuses, possibly producing negative pressure, local treatment is advised. This usually consists of a nasal spray of 3 per cent ephedrin sulphate about the middle turbinate every two or three hours, followed by hot mineral oil or an ointment. Hot saline irrigation, suction, tampons of argyrol, etc., all tend to assist in the establishment of the ventilation and drainage of the posterior sinuses, and are advocated as the appropriate method for office treatment. The only surgical procedure indicated in most cases is the removal of the focus. In a few, it is good judgment to also remove nasal obstruction when well marked, such as deflections

of the septum or hypertrophy of the middle turbinates, conditions that usually need correction, irrespective of the eye disturbance.

For a few of the most serious cases, where optic atrophy is a real menace, I have always advocated the opening of the sphenoid¹² and possibly the posterior ethmoid cell in addition to the removal of the focus and nasal obstruction if present. These cases usually have complete loss of vision and small optic canals.¹³ This procedure is done as a therapeutic measure, and the benefits come from ventilation and depletion of the structures adjacent to the optic nerve and not from removal of any focus of infection. The percentages of these cases have from year to year become lower and may eventually vanish. This percentage is now somewhat under 10. In the thirty cases appended to this paper the sphenoids were opened but twice, and in neither case was it justifiable, as both would undoubtedly have recovered simply by the removal of the focus. With our present comprehension of this subject we would not now open either of the sphenoids.

The only ethmoid operation was the removal of enough of the anterior cells to facilitate drainage of an acute frontal sinusitis.

The frontal was opened in but one case in conjunction with the removal of infected teeth and tonsils.

The antrum was opened in four cases. In none of these was it the sole focus.

Thus the sinuses in these thirty cases played but an insignificant rôle.

The location of the focus in the thirty cases was as follows:

Teeth.....	9
Teeth and tonsils.....	3
Teeth, tonsils and antrum.....	1
Teeth, tonsils and frontal.....	1
Tonsils.....	10
Tonsils and antrum.....	2
Pansinusitis.....	1
Influenza.....	1
Acoustic neuroma.....	1
Acute coryza.....	1

Various operations were performed on twenty-seven, with the following results:

In 8 cases the extraction of one or more infected teeth was followed by normal vision in 5, marked improvement in 2 and no improvement in 1 (optic atrophy).

In 3 cases the removal of the tonsils was followed by normal vision in 2, improvement in 1.

Infected teeth and tonsils were removed in 4, followed by normal vision in 3, no improvement in 1 (optic atrophy).

Further loss of vision was prevented in a patient with optic atrophy by removing the teeth and tonsils and opening the frontal sinus.

Resection of the septum and removal of the middle turbinates restored the vision in 2.

Normal vision in two others followed the resection of the septum and removal of the middle turbinates and tonsils.

Normal vision in another case followed resection of the septum, removal of middle turbinates and opening the sphenoid.

The sphenoid was opened in but one other case from which infected tonsils were removed, with resulting normal vision.

No improvement followed the resection of the septum and opening of the antrum of a chronic case.

The removal of the tonsils and middle turbinates was likewise unsuccessful in another chronic case.

The removal of teeth and tonsils and opening the antrum improved one case, while another was benefited by removing the tonsils and opening the antrum.

In but one case (the patient with pansinusitis) were any of the ethmoids involved. In this case the middle turbinate was removed, and the antrum and anterior ethmoids opened. Normal vision was obtained.

Vision in all the acute cases was either restored or greatly improved.

There were but five cases undergoing operation which did not improve. These had either marked optic atrophy or were of such long duration that the operation was undertaken, not with any expectation of improvement, but simply to prevent further loss of vision. It usually succeeded in accomplishing this.

One of the three cases which was not operated upon was hopeless, as far as restoration of vision was concerned, and no operation was advised. The second case neglected to have anything done until too late. The third case had an acoustic neuroma, probably in an operable stage, and although referred to Dr. Cushing, failed to go.

G. K., 40, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on November 24, 1924, with diagnosis of optic neuritis, right.

History: Good general health. No previous eye trouble. Pain and lameness of right eye for ten days. A week ago the vision began to diminish rapidly.

Nasal examination: High deviation of septum to left with considerable irregularity throughout. The right middle turbinate is hypertrophied but there is not complete blocking. Tonsils injected and probably infected. Pyorrheal pockets about teeth.

X-ray report: "Sella normal, sinuses negative. Optic canals normal in size."

Neurologic report: Nothing was found in the neural examination to account for the neuritis. The Wassermann was negative.

Eye findings: The right disc was injected. The edges were indistinct. The fields greatly contracted and blind spot enlarged. Vision o. d. 10/200, o. s. 20/30.

Operation advised.

The neuritis was thought to be due to the infection in the tonsils and their removal was advised. An appointment was made for this operation but the patient failed to return to the Infirmary although the Social Service Department endeavored to have him do so. On February 27, 1926, he did return in reply to a follow-up letter and I learned that after some weeks' delay he had the tonsils removed together with both middle turbinates and that both sphenoids were also opened. The eye findings on that date was "right disc pale, secondary atrophy. Margins somewhat blurred and irregular. Retinal surroundings show remnants of old exudate. Arteries very small, veins about half normal size. Left disc normal. Vision o. d. 20/200 unimproved, o. s. 20/20-2.

Comment: This case interested me greatly as when I first saw him the neuritis was of but ten days' duration and a favorable outcome should have followed the removal of the tonsils. In spite of excellent co-operation on the part of the Social Service the patient refused to return and although he was somewhat later thoroughly cleaned up by an outside specialist, there was no improvement in the vision. Probably the delay in removing the focus was responsible for the marked optic atrophy. A minor operation done in the early stages would probably have accomplished what a much more radical one weeks later failed to do. The removal of the middle turbinates and the opening of the sphenoids was not advised and, as it proved, did not benefit the case.

E. H., 31, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on November 28, 1924, with diagnosis of bilateral optic atrophy. This case has been under observation on the eye side for five years so the history is most instructive.

Vision in 1919 was ou 20/30.
1921 20/40.
1922 20/30.

while at present it has dropped to 20/200.

Examination: Septum straight. Turbinates essentially normal, no blocking. The tonsils have been removed.

Eye report: Both discs show marked pallor. Vision ou 20/200.

X-ray report: Four abscessed teeth. Both antrums contain thickened membrane. The other sinuses are negative. Sella and optic canals normal.

Neurologic report from Massachusetts General Hospital: "Blood, spinal fluid and Wassermann negative. No pathology found in spinal fluid, no signs of organic disease except 2nd nerve. We find no cause for optic atrophy."

Operation: The four abscessed teeth were extracted.

On December 15, 1925, the vision was 20/200 and the general health was somewhat improved.

Comment: This case was, of course, hopeless as far as any improvement in the vision was concerned. The extraction of the four abscessed teeth has apparently stayed further deterioration of the vision, at least it has remained stationary for over a year. Had the importance of studying his teeth been appreciated when he was first examined in 1919, much of the subsequent loss might have been prevented.

A. B., 20, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on November 29, 1924, with diagnosis of retrobulbar neuritis, right, of two weeks' duration.

History: The loss of vision was preceded by pain about the eye for two days; no recent colds.

Examination: High deviation of septum with crowding of right middle turbinate. Tonsils submerged, glands below angle of jaw both sides. Teeth good.

X-ray report: "Frontals and ethmoids clear, both antrums, especially left, show thickened membrane but no retained secretion. Optic canals 5 x 6 mm. Sella normal."

Eye examination: Fundi normal, right pupil larger than left and reacts sluggishly to light but promptly to consensual reaction. Vision o. d. fingers at 15 feet, o. s. 20/30."

Ten days after the removal of the tonsils and adenoids the vision was 20/20 ou.

Comment: This case was probably of tonsillar origin and while the nose showed some obstruction and both antrums contained thickened membrane, recovery followed so soon after the removal of infected tonsils that intranasal surgery was entirely uncalled for.

G. H. P., 49, was referred by Dr. David Wells on December 12, 1924, with tentative diagnosis of bilateral optic neuritis.

History: General health rather poor. Has been subject to colds all his life. Generalized headaches for past year which have been getting progressively worse. Feels unbalanced when looking quickly from one object to another. Has been losing hearing in left ear for a year or more and for past six months says he has been absolutely deaf on that side. There has been considerable tinnitus on the left. The tonsils were removed a year ago. Vision has been failing in left eye for seven months. Thinks left eye has been a little wider open than the right for three months. He considers the right eye normal. There has been a sensation of numbness on the left side of the face for some months. Two weeks ago the vision of the left eye became much worse. Everything seemed blurred. This condition has been getting worse daily. Has been having a cold for past two weeks.

Examination: Septum irregular with ridge on left and general deflection to right. Turbinates about normal, no blocking. Tonsils well removed. Chronic pharyngitis. Considerable tenacious mucus on posterior wall of pharynx. The hearing in the right ear was normal. In the left a watch was not heard on contact. A loud voice could only be made out close to ear. The lower limit was 1024 v. s., Galton at 3 and bone conduction was cut down to one-half the normal.

X-ray report: Sinuses show no evidence of pathology. Sella normal. Optic canals 5.5 mm. Skull shows no evidence of increased intracranial pressure. Teeth negative.

Eye findings: There is swelling of the left disc of about 2 diopters with extensive hemorrhages. The right disc also shows some swelling but no hemorrhages. Vision o. d. 16/30, o. s. 16/200.

Advice: The marked loss of hearing with diminished bone condition, numbness of the left side of the face and papilledema all pointed to an acoustic neuroma on the left side. I reported my findings to Dr. Wells and advised him to refer the patient to Dr. Harvey Cushing, as it seemed a favorable case for surgical intervention. Dr. Wells did as I advised but acting on other advice the patient did not go to Dr. Cushing but to another institution where elaborate tests were made. Pituitary disease was suspected and an operation attempted, probably a subtemporal approach to the pituitary. Dr. Wells sent me the autopsy report some four months later and congratulated me on my correct diagnosis. The autopsy revealed "a neurofibroma probably originating from the auditory nerve."

Comment: Although I have been looking for acoustic neuromas since Dr. Cushing's books on this subject appeared some years ago, this is the only case I have found. It is to be regretted that Dr. Cushing could not have had an opportunity to examine this case and possibly remove the neuroma.

M. H., 17, was referred by Dr. Ralph Hatch on December 13, 1924, with diagnosis of retrobulbar neuritis, right.

History: No previous eye trouble. Right eye became lame and sensitive to pressure six days ago following a severe cold. There has

been a thick mucopurulent discharge from right side of nose for a week. Vision has been failing since trouble started. Tonsils were removed eleven years ago. Frequent bilateral headaches, worse in afternoons.

Examination: Transillumination of antrum negative. Very large right middle turbinate which is wedged between ethmoid wall and septum. Septum thickened but fairly straight. Tonsils well removed. Teeth do not suggest pathology.

Eye findings: Fundus negative. The visual field of right eye shows a small area round fixation point where she has vision. Vision 20/200. X-ray report: "Sinuses negative. Sella normal. Optic canals 5 mm."

Operation: Septum resected. Right middle turbinate removed and the right sphenoid opened. Mucous membrane in region of sphenoid apparently thickened and inflamed.

Vision rapidly returned to normal within a month. Following a severe cold a year later the same eye became sensitive and the vision was somewhat reduced but recovery followed local treatment.

Comment: This was a rather mild case. The obstructing middle turbinate probably produced a blocking to ventilation and drainage of the posterior sinuses. Recovery would probably have taken place had the sphenoid not been opened. This is not now done in similar cases. The mild recurrence would seem to indicate that the nerve was unusually vulnerable. There may be some focus that has not been yet discovered.

C. H., 32, was referred by Drs. F. C. Spalding and George Badger on February 20, 1925, with diagnosis of bilateral retrobulbar neuritis.

History: The patient was recovering from the influenza and her general health was very poor. Had a sore throat with the influenza. No previous eye trouble. Present trouble commenced a week ago with severe headache and vertigo. The eyes were lame and sensitive to pressure. Within 48 hours she lost all her vision. At present sees objects indistinctly. The headaches and vertigo continue. Has had some enlargement of the cervical glands for three months.

Nasal examination: The nose was negative. There was no blocking. The tonsils are small but appear diseased. Has marked enlargement of the cervical glands on both sides but somewhat more pronounced on the left. The left drum membrane has been largely destroyed but the ear is dry. Teeth have been extracted.

X-ray report: "Right frontal small, right ethmoids cloudy. Other sinuses clear. Sella rather small. No signs of increased intracranial pressure. Optic canals 5 mm. Both mastoids completely sclerosed. No destruction can be made out beyond left antrum. This appears to contain a cholesteatome."

Eye findings: Fundi normal. Vision large objects. Visual fields contracted.

Operation: As the tonsils were thought to be responsible for the cervical adenitis they were removed on March 23, 1925, a month after the patient came under observation.

Up to that time her general condition did not warrant this operation. The vision was improving slowly under local treatment. The tonsils were sectioned and found to be infected. The vision improved somewhat. The glands were not removed at this time as it was thought that they might disappear after the infection from the tonsils was eliminated. They did not so disappear and removal was advised, but the patient was forced to return to England before this could be done, and I referred her to Dr. St. Clair Thomson. The vision when she left me was about 20/100 in each eye. Dr. Edward D. Davis of London saw the patient for Dr. Thomson and wrote me as follows:

"I investigated her case very thoroughly and could find nothing to account for the retrobulbar neuritis beyond the fact that the glands in the neck are tuberculous, and I removed one gland under a local anesthesia for pathological examination, and the pathologist gave a definite decision that it was tuberculous. In addition a blood count shows a definite diminution in the leucocytes. I have often suspected tuberculosis as a possible cause of toxic retrobulbar neuritis, so called, but have never been able to prove it. At any rate, the only thing we could find amiss with Miss H. was a lymphatic tuberculosis. Certainly there was nothing in her nose to even justify an exploratory operation."

The tuberculous glands were removed some five months later but there was little if any improvement in the vision. The general health was benefited but she was still in a sanatorium.

On February 24, 1927, Dr. St. Clair Thomson again reported on the case:

"On receipt of your letter I again communicated with Dr. E. D. Davis of Charing Cross Hospital, who writes me as follows: 'The condition of her eyes remains the same. A definite diagnosis of disseminated sclerosis was made. . . . Repeated nose and throat examinations did not reveal any cause for neuritis.'"

Dr. Thomson then goes on to say: "The recognition of disseminated sclerosis appears to be becoming more frequent. Physicians are given to suspect it in those cases of retrobulbar neuritis where the sinuses are negative."

Comment: This case is most unique. The retrobulbar neuritis was at first attributed to tonsillar infection, but when the tubercular glands were removed Dr. Davis thought it was due to the tuberculosis, but later developments have shown the case was one of disseminated sclerosis. Possibly the infection in the tonsils and glands may have been behind the sclerosis.

G. M., 50, referred by Dr. C. F. Worthen on February 26, 1925, with diagnosis of retrobulbar neuritis, right.

History: Good general health. Has had colds about all winter but no sore throats. No headaches. Two months ago noticed that he could not see the top of letters or objects with right eye. No sensation of soreness or lameness in either eye. The loss of vision has been gradual. Is a moderate smoker and formerly drank somewhat.

Examination: Left side of nose normal. Slight deflection of septum to right, slight blocking. Tonsils small but suggest pathology.

Several suspicious teeth. Enlarged cervical glands below angle of jaw, more marked on right.

Eye findings: Slight pallor of nerve on temporal side. No change in visual fields. Vision fingers at 10 feet right, 20/70 left.

X-ray report: "Sinuses show no condition of pathology. Right optic canal definitely smaller than left (right 4 mm., left 5 mm.) Sella normal. Upper left lateral incisor abscessed. Lower left bicuspid abscessed. Pyorrheal pockets about lower right molar and upper right molar, also about lower left second molar and upper left bicuspid."

Operation: The patient had the abscessed teeth extracted and the pyorrheal pockets cleaned out. Vision improved slowly but was practically normal when he was checked up a year later.

Comment: The marked dental infection so overshadowed everything else that it was not thought necessary to ventilate the posterior sinuses, although this might have been considered from the size of the optic canal had the case been more acute.

S. C., 35, was referred by Dr. A. L. Macmillan, Jr., of Concord, N. H., on April 15, 1925, with diagnosis of retrobulbar neuritis, right.

History: Fair general health but overworked. Has violent headaches. Smokes one pipe a day. Had influenza three weeks ago. The left eye has been poor since childhood. One week ago the right eye became foggy and within three days dropped to 2/200. The eye was lame and sensitive.

Examination: Right antrum darker than left on transillumination. Marked deflection of septum to right. Both middle turbinates hypertrophied and there is marked blocking on both sides. The tonsils are ragged and congested and the crypts contain offensive secretion, but there is no involvement of cervical glands.

X-ray report: "Both optic canals large. Sinuses show no evidence of pathology. Sella normal. Upper right and left third molars unerupted but not impacted."

Physical and neurologic examinations negative.

Eye report: Both fundi normal. Vision o. d. 2/200, o. s. 20/200.

Operation: The septum was resected and both middle turbinates removed.

The vision returned rapidly. Within a month it was 20/40 and in two months practically normal.

Comment: In this case it was felt that the influenza was responsible for the neuritis and although the tonsils were suspicious it was not considered advisable to remove them at the first operation. Had the vision not promptly improved they would have been removed later. The sinuses being all negative, were not opened. Relief from the blocking seemed to be all that was necessary.

D. M., 35, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on April 16, 1925, with diagnosis of bilateral optic atrophy.

History: Good general health. Uses cider in moderation but tobacco excessively. Six months ago he suddenly lost the vision in both

eyes. Did not have a cold at the time. He thinks the vision has improved slightly.

Examination: Septum deviated to right and in firm contact with a hypertrophied right middle turbinate. There is a large polyp on the left and a degenerated middle turbinate. Tonsils small and submerged. No glands found. Many carious teeth.

X-ray report: "Left frontal sinus definitely pathologic. Considerable reaction in the bone about sinus. Rest of sinuses clear. Optic canals and sella normal. Left lower second molar roots and upper left first bicuspid abscessed. Upper left molar roots infected."

Eye findings: Fundi show marked pallor on temporal side of both discs. Has central scotoma. Both eyes detect blue but miscall red and green.

Operation: On May 19, 1925, left Killian. Tonsillectomy and extraction of abscessed teeth.

October 29, 1925: Vision o. u. fingers 5 feet.

March 16, 1926: Vision o. u. fingers 10 feet, discs pearly white.

Comment: There was marked optic atrophy when this patient was first examined. The infection in the teeth and left frontal was sufficient to cause this. The operation prevented further loss, in fact, there was a slight gain, but permanent damage to the nerve had taken place. This case emphasizes the necessity for early and prompt removal of the foci whenever we are dealing with a severe optic nerve lesion.

B. W., 42, referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on April 23, 1925, with diagnosis of retrobulbar neuritis, right.

History: Patient has had defective vision in the left eye since childhood when the globe was injured by a blow, with resulting opacity of the cornea. Has been losing vision in the right eye for past six months. Has had frontal headache in the forenoon but subsiding in the afternoon. Also some vertigo.

Examination: The septum was irregular and both middle and inferior turbinates somewhat enlarged. Tonsils suspicious.

X-ray report: "Both antrums definitely pathologic, showing thickened membrane. Ethmoids also show some thickening. Optic canals normal in size. Sella normal."

Eye findings: Fundus shows nothing definite. Vision o. d. 1/100, o. s. fingers 1 foot.

Operation: The septum was resected and both antrums opened intranasally. The right contained a large amount of pus and polyps, the left only thickened membrane.

The patient was advised to have his tonsils removed but neglected to do so. When seen nine months later the vision was unchanged.

Comment: The prognosis was poor as the loss of vision was of six months' duration. There was enough pathology in the antrums to warrant the operation irrespective of the eye condition. The patient was not cooperative, became discouraged and neglected to report for a tonsillectomy. This case illustrates the necessity for early removal

of the source of infection and emphasizes the fact that irreparable changes take place in the nerve when this is neglected.

P. T., 40, referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on April 23, 1925, with diagnosis of bilateral retrobulbar neuritis.

History: For past year vision has been slightly defective. He has also had severe occipital headaches and quite marked vertigo. The headaches come on mornings and clear up afternoons. Vision seems to be affected by changes in the weather. Has been under treatment in the eye clinic for six months without improvement. At first a diagnosis of toxic amblyopia was made as the patient was an excessive smoker and drank considerable beer but there was no improvement following discontinuance.

Examination: High deviation of septum to left with marked thickening. Middle turbinate touches septum, the right for its entire length, the left anteriorly. Tonsils diseased. Glands on both sides. Several carious teeth.

X-ray report: "Left antrum contains pus. Left second molar abscessed. Optic canals and sella normal."

Eye report: Papillomacula bundle, o. u. definitely pale, central scotoma for red and green. Vision o. d. 20/30, o. s. 20/30-3.

Operation: The tonsils and abscessed tooth were removed and a radical operation done on the left antrum.

Following the removal of the various foci the headaches and vertigo were relieved and the vision stabilized, i. e., on February 20, 1926, it was o. u. 20/30.

Comment: Some permanent damage to the nerve had taken place but further atrophy should be checked by the removal of such definite foci. I did not think it as necessary to ventilate the nose as to remove these foci and this was all the patient was willing to have done. Removal of the nasal blocking was advised.

F. L., 53, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on June 3, 1925, with diagnosis of bilateral retrobulbar neuritis.

History: Fair general health. Is not subject to colds or sore throats. Uses alcohol and tobacco to excess. Had sudden bilateral loss of vision three months ago. This improved somewhat but six weeks later the vision suddenly became poor again and has so remained.

Examination: Hypertrophy of the right middle turbinate with **marked blocking**. Submerged tonsils. Cervical gland right. Teeth have been extracted.

X-ray report: Sinuses negative, sella normal. Optic canals 4.5 x 5 mm., increased density of periphery.

Eye findings: Beginning atrophy temporal side, both discs pale. Central color scotoma. Vision 15/200 not improved.

Massachusetts General Hospital report: Wassermann negative. No organic nerve lesion found.

Operation: The tonsils and right middle turbinate were removed.

On October 7, 1925, the vision was unchanged.

March 23, 1926, the vision was o. u. fingers at 12 feet. Still uses alcohol to excess.

Comment: There is considerable question in this case as to etiology. If the infection was in the tonsils, as was thought probable, the atrophy had progressed so far that their late removal was without benefit. The persistent use of alcohol of course might have produced the atrophy.

J. S., 36, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on June 20, 1925, with diagnosis of bilateral retrobulbar neuritis.

History: Good general health. Rather frequent colds. Uses alcohol and tobacco to excess. Sudden bilateral loss of vision three weeks ago, which was preceded by a severe cold. No previous eye trouble.

Examination: Both middle turbinates hypertrophied and in contact with septum, which is thick and irregular. Tonsils appeared to be diseased. Teeth suspicious.

X-ray report: Sinuses negative. Sella and optic canals normal. Teeth negative.

Eye findings: Fundi negative. Central color scotoma for red and green. Vision o. u. 15/200. Owing to the history of excessive use of alcohol and tobacco it was thought the condition might possibly be a toxic amblyopia.

Operation: On June 27th the tonsils were removed and the septum resected. This was followed by marked improvement in vision.

Comment: In this case the infection in the tonsils warranted their removal even if the case was really one of toxic amblyopia. The history of a recent coryza with probably an acute flareup of the tonsils favored this view.

V. R., 29, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on July 11, 1925, with diagnosis of bilateral retrobulbar neuritis.

History: Good general health. Has not been subject to colds or sore throats. Drinks four bottles of wine and smokes several cigars per day, so it was thought possible that his present trouble might be from alcohol or tobacco. There has been gradual loss of vision for three or four months. At no time have the eyes been sensitive to pressure or lame.

Examination: Nose negative. Hypertrophied tonsils but no enlargement of the cervical glands. The teeth do not suggest infection.

X-ray report: "Thickened membrane of the left antrum. Sella and optic canals normal. Upper left canine unerupted and malposed."

Eye findings: "Fundus show some fine vitreous opacities. Lamina cribrosa seen plainly in center of discs. Discs not pale. Vision o. d. 20/200, o. s. 20/100."

Neurologic and Wassermann examination negative.

Operation: On June 24, 1925, a left radical antrum and tonsillectomy was done. The vision improved considerably. Four months later it was o. d. 20/50, o. s. 20/30.

Comment: This was a case in which there was some doubt as to the diagnosis. The infection in tonsils and antrum seemed sufficient to warrant removal and marked benefit followed this procedure.

H. D. H., 9, was referred by Dr. Walter B. Lancaster on August 3, 1925, with diagnosis of retrobulbar neuritis, left. As I was on my vacation the case was followed by Dr. Leon E. White, Jr.

History: Good general health. Had acute nephritis four years ago but this cleared up. Sixteen days ago had pain over right eye with subsequent loss of vision in left.

Examination: High deviation of septum to right with hypertrophy and crowding of left middle turbinate. Tonsils diseased.

X-ray report: "Sinuses all clear except left ethmoids which are very slightly thickened. There is no evidence of retained secretion. The sphenoid is perfectly clear. The optic canals are within normal limits."

Neurologic examination was essentially negative.

Eye findings: Dr. Lancaster reported that in November, 1924, the patient had a small refractive error, not sufficient to account for his backwardness at school. No glasses were advised. Fundus normal. July, 1925, well marked papilledema. Fields show concentric contraction left and slight relative central scotoma. Vision left 6/30, right 6/8. August 5th, right eye still 6/8, but showed some concentric contraction and enlargement of blind spot and slight relative central scotoma.

Operation: Left middle turbinate removed, membrane red. Sphenoid opened, membrane normal. Tonsillectomy.

Result: Dr. Lancaster reported "the vision in the left eye returned to 6/6—; central scotoma disappeared; the right eye was 6/5, fields restored." When Dr. Lancaster saw the patient in November, 1926, the "left optic disc showed limited area of whiteness not well marked. Vision normal."

Comment: This was an acute case of short duration, probably from tonsillar infection and possibly their removal alone might have sufficed. In commenting on the case Dr. Lancaster said it was "a case of inflammation affecting the optic nerve not exclusively retrobulbar, but both retrobulbar (as shown by the central scotoma) and intraocular (as shown by the papilledema, the enlarged blind spots and the contracted fields)."

M. C., 30, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on August 5, 1925, with question of retrobulbar neuritis, right.

History: Six weeks ago had severe temporal pain and three days later became blind in right eye. Pain has continued. No previous eye trouble. General health good. Had a sore throat a year ago.

Examination: High deviation of septum to right touching right middle turbinate which is not hypertrophied. Tonsils diseased.

X-ray report: "Sinuses negative, teeth negative."

Eye findings: "Slight light perception on temporal side of right eye. Right pupil slightly larger than left. Has paresis of right external rectus muscle. Vision o. d. nil, o. s. 20/20, fundi normal."

Massachusetts General Hospital reported "Wassermann strongly positive. X-ray shows skull negative. Discharge diagnosis: Aneurysm of right internal carotid."

Dr. Gilbert Horrax of the Peter Bent Brigham Hospital examined the case but was unable to find any neurologic explanation for the amblyopia.

Comment: The sudden unilateral loss of vision with a negative fundus strongly suggests a retrobulbar neuritis. The positive Wassermann would lead one to the belief that the leptic infection was in some way responsible. As to the grounds for diagnosing the loss of vision as due to aneurysm of the internal carotid I do not quite see the necessity for this unless on something more definite than theory. I am looking for further data.

H. R., 46, was referred from the eye clinic of the Massachusetts Eye and Ear Infirmary on August 20, 1925, with diagnosis of bilateral retrobulbar neuritis.

History: Poor general health. No previous eye trouble. Four months ago the patient suddenly lost most of the vision in both eyes. There does not seem to have been any change since the trouble commenced. Everything seems to be blurry. He did not have a cold or headache.

Examination: Anterior deviation of septum. Middle turbinates hypertrophied. Tonsils suspicious. Teeth suggest infection.

X-ray report: "Sinuses negative. Optic canals 4.5×6 mm. Sella normal. Teeth: Lower right first molar, upper right and left central incisor and upper left lateral incisor all abscessed."

Eye findings: Fundi normal. Central scotoma for red and green. Vision o. u. 20/200.

Neurologic examination: Toxic neuritis, possibly alcoholic. History of alcohol. Dental infection may account for source.

Operation: Abscessed teeth extracted.

Result: February 27, 1926, the patient returned for check-up. His general health has greatly improved; has gained 20 pounds. Vision o. d. 20/40—1, o. s. 20/50—2.

Comment: The result, considering the four months' duration, was unusually good. The teeth were probably the principal source of the neuritis. There did not seem to be any occasion for opening the posterior sinuses, nor do I believe that a better result would have followed such a procedure. The suspicious tonsils would have been removed had not improvement followed the extraction of the teeth.

L. S. B., 24, was referred by Dr. Allen Greenwood on September 5, 1925, with diagnosis of retrobulbar neuritis, left. As I was on a vacation, this case was handled by my son and the following notes were taken from his record:

History: Good general health. Tonsils removed about a year ago. Has had a full feeling in right eye about three months. The trouble in the left eye commenced a month ago with pain and blurring of vision. Had a similar trouble a year ago but not as severe. This cleared up after removal of the tonsils. Patient also has an acute secretory left ear.

Examination: High deviation of septum to right with hypertrophy of both middle turbinates. Diseased tonsil stumps.

X-ray report: "Sinuses show no evidence of pathology. Both optic canals are rather small, right 4×5 , left 5.5×4.5 mm."

Eye findings: Dr. Greenwood reported that a year ago there was partial loss of vision in right eye. This gradually improved without any special treatment. A month ago the left eye started to pain and the vision was cloudy. Right eye had, with proper glass, vision 20/30. The nerve head in this eye was rather white, suggesting an old retrobulbar neuritis; the field was normal. The left eye with proper glass had a vision of 20/40 seen through a cloud. The fundus in this eye was normal but there was a rather vague central relative scotoma for form and colors. Diagnosis made of old retrobulbar neuritis, right, and fresh retrobulbar neuritis, left. Six weeks after first examination (that is, five weeks after operation) vision both eyes 20/20 with proper glass.

Operation: Submucous resection, both middle turbinates removed and tonsil stumps.

Result: Vision improved rapidly for two weeks, then patient had several severe nasal hemorrhages that required packing for some days. During this period vision dropped to where it was before operation. When packs were finally removed there was a slow return of vision. Five weeks after operation it was normal in both eyes. The vision is still unstable, especially when the patient is tired or has a cold, otherwise it is normal.

Comment: The optic canals, especially the left, are small and I think the sphenoid should have been opened. I have seen the patient in consultation with my son and he agreed that should this instability of vision persist, the left sphenoid should be ventilated. The hemorrhages following the operation have so demoralized the patient that she will not consent to anything surgical unless the eye troubles more than it does at present.

V. B. M., 31, was referred by Dr. Alexander Quackenboss on October 5, 1925, with diagnosis of retrobulbar neuritis, right.

History: Fair health but rather tired and somewhat under weight. Has had arthritis. Is not subject to colds. Has occasional sore throats. A week ago on awakening discovered that there was a "shadow" over the right eye and the vision has failed so that at present it is nil. The eye is sensitive and lame.

Examination: Septum thick and irregular. Middle turbinates crowded between septum and outer wall. The right side is somewhat obstructed. Tonsils of moderate size, crypts contain offensive secretion. Some congestion of anterior pillars. Several suspicious teeth.

X-ray report: "Sinuses show no evidence of pathology. Entire sphenoid bone very pneumatic. Air cells go into lesser wing right and none left. Posterior clinoidal process very thin and arched over to meet the anterior. No evidence of pituitary disease. Right optic canal 4.5 mm., left 5 mm. There are four abscessed teeth."

Eye report (Dr. Quackenboss): "Patient called September 29, 1925; at that time the vision in the right eye was two-thirds of normal, the field of vision was normal and I could make out no central scotoma. The fundus was normal; the eye was tender on motion and a diagnosis of retrobulbar neuritis was made. The following day the vision was reduced to one-twentieth of normal and the edge of the optic disc was slightly blurred. She telephoned later that the vision in the eye was worse but I did not see her until October 13th, when the vision in the eye was normal, the visual field was good, the optic disc showed a mild neuritis, the edges being indistinct."

Treatment: Epinine ointment at home. Argyrol and adrenalin spray followed by hot saline irrigation in office. Referred to dentist for extraction of teeth.

Results: Following the removal of the abscessed teeth the vision rapidly improved and was normal within a month.

Comment: The abscessed teeth was so evidently the cause of the neuritis that the tonsils, although suspicious, were not removed. Had the vision not improved promptly this would have been indicated. The obstruction in the nose was so slight that ventilation was obtained by treatment. The prompt recovery shows how little is needed in this type of case except the removal of the focus.

C. M. R., 27, was referred by Dr. David Wells on October 24, 1925, with diagnosis of neuroretinitis, left.

History: Fair general health. Is subject to colds and sore throats. The left side of nose has always been somewhat obstructed. Had a trouble similar to the present eye difficulty in March, 1918, and the Infirmary record states that there was "marked papillitis O. S., veins very prominent and large in comparison with arteries. Injected condition of retina. No hemorrhages, discemitis O. S. and vitreous opacities." Present attack commenced a week ago with specks in the left eye and it now feels as if it would drop out.

Examination: The left middle turbinate was enlarged and in contact with septum and outer wall, thus producing considerable blocking. The tonsils were submerged and there was some congestion of the anterior pillars. There were several suspicious teeth.

X-ray report: "Sinuses show no evidence of pathology. Sphenoid is quite pneumatic and the pneumatization extends into the lesser wing. The left optic canal is smaller than the right, right 5.5 mm., left 4.5 x 5.5 mm. There is an area of rarefaction between upper left lateral incisor and central incisor. End of root itself appears softened. Well to consider its removal."

Eye findings: Dr. Wells reported October 23, 1925: "Nerve not swelled more than the surrounding tissue but is infiltrated, rather an

opaque look. The blood vessels are not specially tortuous. There is considerable haziness of the vitreous so that the whole fundus is somewhat dull, also a fine granular deposit in the cornea. Vision today 16/70." Dr. Wells reported a week later: "Fully as much inflammation of the nerve. Thinks vision slightly improved. Blind spot slightly enlarged but no central or paracentral scotomata. Evident infiltration of anterior layer of cornea."

Dr. Edward N. Libby made a careful physical examination and reported no evidence of focal infection in any part of the body.

Dr. L. M. S. Miner reported as follows on the teeth: "No deep seated infection." One suspicious tooth had previously been removed but the eye condition did not improve.

The tonsils were the only probable remaining focus and I removed these on November 11th. They appeared infected.

Two weeks after the operation Dr. Wells reported: "Left eye remains about the same. Nerve is still foggy. There is an opacity in cornea and some haziness in vitreous." A month later he reported: Vision, left 16/40, no new lesion. Left optic nerve continues hazy. Blind spot slightly enlarged."

The eye from then on improved rapidly, so that the vision was soon slightly better in the affected eye than in the other one.

Comment: This was an exceedingly obstinate case, even after the removal of the focus in the tonsils. This was due in a measure to the inflammatory process in the retina and cornea. The final recovery, however, was rapid. The general health was likewise markedly benefited.

M. M., 16, was referred by Dr. Henry Hawkins on December 15, 1925, with diagnosis of retrobulbar neuritis, left.

History: General health was fair, not subject to colds or sore throats, no previous eye trouble. Three days ago the left eye commenced to water and became blurry. It was lame and sensitive. Vision failed rapidly and only finger movements could be made out.

Examination: The septum was markedly deflected to the left. The left middle turbinate was tightly wedged between septum and outer wall. The posterior sinuses were badly blocked. Tonsils were submerged and probably infected, offensive secretion in crypts, and congestion of anterior pillars. The teeth appeared healthy.

X-ray report: "Sinuses show no evidence of pathology. Left optic canal slightly smaller than right. Right 5.5 mm., left 5 mm. The teeth show no evidence of infection."

Eye findings. Fundus negative. Vision finger movements left, 20/15 right.

Operation: The septum was resected. The middle turbinate and tonsils removed.

Pathologic report on tonsils. Marked hyperplasia, mycosis and keratosis of tonsils.

Vision returned rapidly. Within a week fingers could be counted at 20 feet, and in two weeks it was 20/30 plus. In a month it was normal.

Comment: This case made an ideal recovery. There was little doubt as to the focus and no question as to the necessity for removing the blocking in the nose. The opening of any of the accessory sinuses would have been superfluous to say the least.

T. V., 16, was referred by Dr. George Ryder on January 15, 1926, with diagnosis of retrobulbar neuritis, right.

History: Good general health. Had tonsils and adenoids removed a year ago. Previous to that frequent abscesses in his ears. During past winter has had several colds with some blocking of the eustachian tubes. No previous eye trouble. Ten days ago noticed a slight blurring of the right eye. It was sensitive to light but not lame. Has been a little dizzy in the morning and has had considerable pain above the other eye.

Examination: Marked deflection of the septum to the right high up and far back. The right middle turbinate was tightly wedged between septum and outer wall, thus blocking the posterior sinuses on the right. The left middle turbinate was large and degenerated. The tonsils were well removed. The teeth did not suggest infection.

X-ray report: "Left pansinusitis clearing. Frontal and antrum have markedly thickened membrane but no retained secretion. Sphenoid very large and there is air in the anterior clinoid process. The optic canals are oval, left smaller than right; right 4.5 x 5.5 mm.; left 4 x 5.5 mm. Both are compressed laterally, upper edges indistinct. Pneumatic cells adjacent to both."

Eye findings: The edges of the right disc were blurred, vision 20/50.

Operation: The left antrum was widely opened and found filled by soft edematous tissue. Both middle turbinates were removed and the anterior ethmoids on the left.

Vision improved rapidly. In two weeks it was 20/30 plus and the disc normal.

Comment: The left pansinusitis was probably responsible for the neuritis of the right eye. The marked blocking on the right side demanded relief and this was obtained by removing the middle turbinate. The infection in the frontal cleared up following the removal of the degenerated left middle turbinate and the anterior ethmoid cells. The antrum seemed to demand permanent aeration. The posterior sinuses were not opened and the fact that recovery followed so rapidly proved that this was not necessary.

M. L. F., 35, was referred by Dr. Allen Greenwood on March 1, 1926, with diagnosis of retrobulbar neuritis, right. Rather poor health; possibly some hysteria. No previous eye trouble. Has been having considerable headache for past three weeks. A week ago the right eye began to blur. There seemed to be a heavy mist before it and was painful and lame.

Examination: Nose fairly normal. Tonsils submerged. Some congestion of anterior pillars. Enlarged cervical gland, right. Teeth suspicious.

Eye findings: Fundi normal. Central scotoma right. Central field of vision right blotted out for red and green. Vision right 20/200, left 20/20.

X-ray report: "Small cyst on floor right antrum, otherwise sinuses clear. Optic canals 4 mm. Sella normal. Upper right second molar abscessed; lower right and left third molars unerupted and impacted. Remaining teeth show no definite evidence of abscess formation."

In this report on the teeth the phrase, "no definite evidence of abscess formation," made me suspect that some of the other teeth might harbor infection, so I referred the patient to Dr. K. H. Thoma for further investigation (a wise precaution considering the subsequent course of the case). He reported: "Teeth and X-rays examined and I agree that there is decided infection on the right upper second molar and this tooth should be extracted. Two more questionable teeth, left upper first molar and left lower second bicuspid. These teeth show no infection at the root ends as yet, but have very large cavities going close to the pulp and should receive immediate attention. The impacted third molars I don't think have any bearing on the case because they do not appear to be infected." The abscessed tooth was removed two days later.

In four days the vision was 20/30 and within three weeks normal. A little over a year later on May 3, 1927, there was a recurrence of the neuritis and vision of right eye dropped to 20/200. The teeth were again suspected and three were removed by Dr. Miner before the focus was eliminated. Vision then returned to normal and has so remained. Four months later the patient became very dizzy and was practically confined to her bed for some weeks. Nothing was found in her eyes or intestinal tract to account for the vertigo. It seemed to me to be a neuritis of the vestibular branch of the auditory nerve. I suspected that the teeth were again at the bottom of the patient's difficulty, although this time it affected the auditory rather than the optic nerve. The following report from Dr. Miner confirmed me in my suspicions: "Patient complained of dizziness associated with some discomfort on both right and left sides. Examination of dental area revealed a lower right imbedded wisdom tooth with some absorption of bone around it. The first and second molars are absent and the bone in good condition at site of these teeth. On the left side there was an impacted third molar, impacted quite heavily against the second molar with some absorption of bone over this tooth. Clinical examination revealed inflammation of soft tissues in site of these two teeth with some slight discharge, especially on left side. On October 7th both of these teeth were removed under local anesthesia. There was very little reaction the first two days. On the fourth day she had some pain, some swelling and temperature of 100. Treatment brought these symptoms down and on October 20th she was practically entirely free from trouble. On the 24th she reported that the dizziness had disappeared and the only remaining symptom was some headache."

When checked up on November 1st, 1927, the patient was found to be entirely free from the headache and vertigo and the vision was normal.

She stated that ever since her original eye trouble nearly two years ago there had been great difficulty in focusing the eyes and pain on moving or using them for any length of time, especially by lamplight. This was relieved within a week after the extraction of the impacted teeth.

Comment: This case is most unique. The two attacks of retrobulbar neuritis were promptly relieved by the removal of dental infection. A third infection did not hit the optic nerve but the auditory and was as promptly relieved by the removal of the dental focus. Several other cases have given a history of vertigo which leads one to wonder if the vestibular branch of the auditory nerve does not become more frequently involved in conjunction with these optic nerve lesions than anyone has heretofore suspected.

E. F., 24, was seen in consultation at the Massachusetts Eye and Ear Infirmary on March 10, 1926. Diagnosis, optic neuritis, left, with papilledema.

History: General health good. About three weeks ago noticed that movement of the left eye was painful and within a week a black spot appeared before this eye. She discovered that when looking at two cups she could see but one. Five years ago she had a double extenteration of the ethmoids and a double radical antrum. This was followed by an infection in the left middle ear and mastoid which, however, cleared up after incision of the drum membrane. The tonsils have been removed.

Examination: Septum straight. Offensive secretion with tenacious crusts over ethmoid region on both sides. The upper portion of ethmoid labyrinth and stumps of middle turbinate were not removed. The nose needs cleansing and further study. A large part of the left tonsil was also left and might be a source of infection as the cervical glands on this side are somewhat enlarged and sensitive.

X-ray report: "Both antrums pathologic. Chronic sclerosis of bone. Chest plate negative. Sella normal."

General physical and neurologic examinations reveal nothing to account for eye condition. Area at right apex posteriorly suggestive of pulmonary tuberculosis. Spinal fluid and Wassermann negative.

Eye findings: "Nervehead blurry, much congested. Elevated about 2 diopters. Exudate around it obscuring some of the vessels. Several hemorrhages along inferior temporal artery. Vision 20/20—. Fields show enlarged blind spot and green appears grey."

Operation: The tonsils stumps were removed on March 15, 1926. They appeared pathologic. The papilledema commenced to subside within forty-eight hours.

A month later the margins of the disc could be plainly made out and the condition showed distinct improvement. Vision 20/20. On March 26th (two weeks later) the swelling of the nerve was much less; on April 11th the spinal fluid and Wassermann were negative; and on April 12th there was less choking of the disc.

Comment: This case was of unusual interest as the vision was not impaired except by the scotoma. The edema of the disc was the

unusual feature. While the sinuses had been exenterated they still seemed to harbor infection but the possibility of eliminating this in face of the marked sclerosis of the bone seemed dubious. The left tonsil stump was quite evidently infected and its removal, at least as a preliminary procedure, seemed indicated. If the papilledema had persisted an attempt would have been made to clean up the sinuses.

E. B., 41, was referred by Dr. Allen Greenwood on May 11, 1926, with diagnosis of retrobulbar neuritis, right.

History: Good health up to two weeks ago. Not many colds. No previous eye trouble. Seven days ago noticed right eye began to discharge and things looked cloudy. This came on quickly and has remained the same with vision for fingers at about 3 feet. Eye was lame on movement at first but not at present, although it is still sensitive to pressure. Some frontal headache.

Examination: Right side of face somewhat darker than left on transillumination. Marked deflection of septum to left with compensatory hypertrophy of right middle turbinate. One bad tooth. Small gland each side of angle of jaw. Considerable congestion of palate and anterior pillars. Right middle turbinate wedged tight against outer wall of nose.

Eye findings (Dr. Greenwood): "Fundus practically normal. Possibly slight fullness of retinal veins. Central vision worse than peripheral, i. e., large central scotoma."

X-ray report: "Slight thickening of lining of both antra but no evidence of activity and no retained secretion. Both optic canals large. Sella turcica normal."

The teeth were then filmed. An apical abscess was found.

Operation: The infected tooth was removed.

Result: The neuritis subsided rapidly. Within six days there was marked improvement in the vision, although the central field defect did not entirely clear up for some weeks.

Comment: Although the tonsils were suspicious and there was considerable nasal blocking, the neuritis subsided so quickly after the extraction of the infected tooth that it was quite evident that this was the real focus.

N. C., 32, was referred by Dr. C. F. Worthen on June 26, 1926, with diagnosis of retrobulbar neuritis, right.

History: Good health. Many colds during past six months and headaches. One week ago felt as though a hair was in right eye, and a few hours later discovered that everything seemed blurry when viewed with this eye.

Examination: Septum deflected to right posteriorly. Contact between right middle turbinate and septum but no marked blocking. Granular pharyngitis. Thickening behind palate. Tonsils do not suggest pathology. Cervical glands not palpable.

Eye findings (Dr. Worthen): "Vision O. S. shadows. Fundus negative. Light projection faulty O. D. No lesion in fundus except question of temporal pallor to nerve."

X-ray report: "No evidence of infection in accessory sinuses. Following abscessed teeth: Lower right second bicuspid and both molars; also lower left second bicuspid and first molar. There is an infection about upper left first molar; upper left second bicuspid root is not abscessed although the crown is missing."

Operation: The infected teeth were removed.

Result: Following the extraction of the teeth the vision gradually cleared. On July 6th it was fingers 2 feet; on August 27th, fingers at 7 feet; and it eventually reached 4/70. The temporal pallor noted by Dr. Worthen in his first examination increased, so that at present it has involved the entire right disc. There has been no change in the vision during the past year as shown by a checkup on November 10, 1927.

Comment: This case was unusually severe due to the many infected teeth. When first seen there was commencing optic atrophy. Although there was considerable improvement following the elimination of the numerous foci, the nerve was permanently damaged. The teeth were removed one or two at a time. Possibly it would have been better had all showing infection been extracted at once.

L. R. W., 34, was referred by Dr. Alexander Quackenboss on February 5, 1927, with diagnosis of acute retrobulbar neuritis, right.

History: Good general health, excepting occasional severe headaches. Is not subject to colds or sore throats. Does not smoke or drink. Two weeks ago noticed a blurring of the right eye, at first thought it steam on his right lens. The vision grew rapidly worse so that within a few hours he could not see a person's face, only the form. The blurred vision lasted but two or three days, then cleared up, but came back in a few days so that at present there is marked impairment of vision in the right eye.

Examination: Slight septal deflection to left but no nasal obstruction. Uvula edematous, both anterior pillars congested, offensive secretion removed from tonsil on suction. Cultures of this showed staphylococci and streptococci. Enlarged gland at angle of jaw, right. The tonsils were evidently diseased. Several suspicious teeth.

Eye findings: Vision right 5/200, left 20/20. Fundi normal. Color scotoma right for blue and green.

X-ray report: Sinuses and sella negative. "No conclusive evidence of apical rarefaction involving any of the teeth." I did not like the word "conclusive evidence" in this report, so I referred the patient to Dr. K. H. Thoma, who reported as follows: "There is decided infection on the upper left central and slight infection on one root of the left upper first molar. While the bone change as seen in the X-ray is not very great, I feel that a case like his requires radical treatment and removal of all possible infection."

The physical and neurologic examinations were negative.

Operation: On February 8th I removed the tonsils, which were infected, and Dr. Thoma extracted four teeth, all badly infected. This in spite of the fact that the dental films had been first reported as negative.

Vision at once commenced to improve and within ten days was 5/50. A month later Dr. Quackenboss reported: "Vision, fields and fundi all normal." On November 4, 1927, the patient was checked up. There had been no recurrence of his eye trouble.

R. F. L., 31, was referred by Drs. E. W. Clap and C. F. Worthen on February 25th, 1927, with diagnosis of chronic bilateral retrobulbar neuritis with optic atrophy.

History: Good general health, excepting some discomfort in abdomen. Has had a numb feeling on the right side of face for a month. Has been losing the vision of the left eye for about a year. Had some pain in temporal region for two or three days when it started. There was no history of sudden loss of vision. The vision ten months ago, according to an optometrist, was with correction 20/20 right, 20/100 left. He was referred by the optometrist to an ophthalmologist, who in turn referred him to a rhinologist, who resected his septum. Dental films at that time showed two suspicious teeth but these were not extracted.

Examination: Right antrum slightly darker than left on transillumination. Middle turbinate of moderate size and do not obstruct. Tonsils large and considerable offensive secretion was removed from the crypts by suction. Both anterior pillars congested. Small gland at angle of jaw, right. Teeth show several large fillings. Referring to Dr. K. H. Thoma for investigation of teeth. He reported as follows: "Two root filled teeth on right upper side, the first bicuspid and second bicuspid. First shows evidence of erosion at apex and some infection. The second appears fairly normal. Large pocket connected with right upper first molar which is of infectious origin. Left lower first molar shows large cavity underneath gold crown which should be investigated with reference to pulp infection. Advise extraction of three teeth."

Eye findings (Dr. Clap): "Secondary optic atrophy possibly following a very mild papillitis. Vision o. d. 5/70—1 and o. s. 4/200."

Suspecting from the numbness of the right side of the face, the abdominal discomfort and the slowly progressive loss of vision that we were dealing with either an intracranial neoplasm or multiple sclerosis, I referred the patient to Dr. Gilbert Horrax for neurologic study. He reported as follows: "I may confirm my telephone conversation with you by saying that my diagnosis of this man's case was multiple sclerosis. The positive features on a neurologic examination were:

1. Persistent nystagmus to right and left and upward.
2. Slight bilateral optic atrophy.
3. Slight intention tremor.
4. Ataxia of the left leg.
5. Vague sensory disturbance over both lower legs.
6. Abdominal reflexes absent.
7. Knee jerks and achilles reflexes hyperactive on both sides with slight ankle clonus on the left."

In our telephone conversation I mentioned the infection found in teeth and tonsils and he unhesitatingly advocated their removal on the theory that this infection might possibly be a factor in the etiology of the multiple sclerosis. The teeth and tonsils were accordingly removed, hoping that this might prevent further loss of vision.

In reply to my direct question Dr. Horrax said: "I don't believe anyone could say that multiple sclerosis is caused by focal infection in the teeth or tonsils, but I should certainly think that this is a possibility which ought to be eliminated."

This patient was checked up on November 11, 1927. There was no especial change in the fundi. The pupillary reflexes were present and active. There had been a very slight diminution of the vision—in the right eye from 5/70 to 4/70 and in the left from 4/200 to $3\frac{1}{2}$ /200. The general health seemed somewhat improved.

Dr. H. C. Solomon, who saw the patient during my vacation, suggested that possibly the use of typhoid vaccine intravenously to obtain hyperpyrexia might benefit this patient, and I have asked him to give this treatment a trial.

Comment: This case is very instructive. The loss of vision when he was first seen by an ophthalmologist shortly after the trouble commenced, was attributed to some nasal infection, and the rhinologist to whom he was referred could only find a deflected septum. The resection, of course, was of no material benefit. The possibility of infection in the teeth and tonsils was not seriously considered. Multiple sclerosis was not suspected and I doubt if it would have been possible to diagnose it at that time. When he came to me he had marked optic atrophy and multiple sclerosis was suspected. In the eight and one-half months since the removal of the infection in teeth and tonsils there has been but little change in vision. The elimination of the focus of infection seems to have somewhat checked the progress of the multiple sclerosis. While the possibility of benefiting the patient by hyperpyrexia is very problematic, it seems to me eminently worth trial.

L. H., 50, was referred by Dr. Alexander Quackenboss on March 31, 1927, with diagnosis of acute retrobulbar neuritis, left.

History: Good health. Had intestinal grip a week ago and was confined to her home for two days. Has had severe dizzy headaches the past three months. Change of glasses a month ago relieved this condition, but it returned with greater severity when she had the grip. Three days ago she had difficulty in focusing the eyes and discovered that the vision of the left was greatly reduced. Can see the outline of people but not the face. Does not see top letter on chart when looking straight ahead, but can make it out at two feet when looking sideway.

Examination: Moderate deflection of septum to left with slight crowding of the middle turbinate posteriorly. Compensatory hypertrophy of right middle turbinate. When touching the left middle turbinate patient saw with the affected eye a round disc of lavender color. The tonsils were small and did not appear pathologic. Several suspicious teeth. The patient was referred to Dr. K. H. Thoma for

investigation of the teeth, who reported: "Large number of dead teeth each showing infection and this means extraction. Following should be removed: Right upper lateral first and second bicuspid and second molar. Left upper lateral cuspid and first and second bicuspid. Left lower second bicuspid. Other teeth in lower jaw appear to be in good condition."

Eye findings (Dr. Quackenboss): "Outlines of disc are entirely lost. Markings absent. Veins large and tortuous. No hemorrhage through retina. Little grayish area just below disc. Vision 1/30; three days' duration. Central scotoma."

Believing that the teeth were the cause of this very severe neuritis, their removal was advised. This was accordingly done. The case was treated in the office two or three times a week, principally by ephedrin, hot postnasal irrigation, argyrol and hot mineral oil. Improvement was prompt.

On April 20th (about three weeks later) Dr. Quackenboss reported: "The vision in the left eye has improved to 2/10, there is less disturbance of the optic disc, there are several grayish areas about the disc and a number of smaller spots down and out. The trouble in this case apparently is not confined to the nerve."

Again, on June 3rd (about two months from date of onset), he wrote: "The vision in the left eye is nearly 20/30, the disturbance in the disc is subsiding, the outline is good, a slight scleral ring is clearly seen, retinal blood vessels nearly normal, a few white patches above disc and also down and out from disc."

There has been some further improvement since this last report. When the patient was seen on November 1st the vision was 20/30 plus.

M. B., 40, was referred by Dr. Allen Greenwood on October 10, 1927, with diagnosis of acute retrobulbar neuritis, left.

History: Good general health but subject to laryngitis. Five days ago noticed that things began to look queer and on closing the right eye discovered that she could not see with the left. Two weeks previously she had been refracted and this left eye was found normal. Did not have a cold, nor was the eye lame or sensitive.

Examination: Antrum transillumination was negative. Slight hypertrophy of both middle turbinates but no special obstruction. Tonsils small, pillars and uvula not congested. Suction practically negative. The teeth had several large silver fillings. Dr. K. H. Thoma, who examined them, reported: "I found indication of chronic pulpitis in the left lower first molar caused by a very large cavity under a filling. This condition is recent and quite often an active source of infection. All the remaining teeth are in good condition with the exception of a few cavities and a slight pyorrhea on some of the posterior teeth of the upper jaw."

Eye findings (Dr. Greenwood): "Patient came to see me October 10, 1927, with a vision in the left eye of 20/200 and only the upper half of the letter was seen, owing to a relative scotoma just below fixation and running up to fixation.

Operation: The infected tooth was removed.

This case was treated in the office two or three times a week in the usual way (ephedrin, hot alkaline irrigation, hot mineral oil and argyrol packs). She also used an ephedrin spray at home. Vision was stationary for about ten days, after which the improvement was rapid.

On November 2, 1927, Dr. Greenwood reported: "She has today a vision of 20/30 in the left eye and no scotoma, a very satisfactory cure of the condition, and I expect that ultimately the vision in the left eye will be as good as that in the right eye. The fundus shows nothing abnormal."

The patient is still under observation.

Comment: Dental infection, while practically confined to one tooth, was so evidently the cause of the retrobulbar neuritis that I felt recovery would speedily follow its removal, as it did. This case illustrates how little is needed in the surgical line when one has located the focus.

SUMMARY.

Some focus in the body is responsible for the optic nerve lesion arising from infection. Invasion is through the blood stream. The accessory sinuses supply but an insignificant number of the foci. Most of the foci are found in teeth and tonsils. The sinuses, from their priority of investigation, have occupied the attention of rhinologists too long to the exclusion of other more frequent seats of infection. Much can be learned from the general medical man on the best methods of investigating focal infection. Dental infections are easily overlooked. An expert odontologist is essential in a successful search for dental pathology. Devitalized teeth are a potential source of infection. Tonsils are a frequent source of infection and should be removed when at all suspicious if no other foci are found. The antrum among the sinuses is the most frequent seat of infection. Thickening of the antral mucosa without secretion may be a sufficient focus to warrant surgical intervention. Definite infection in the other sinuses should be eliminated, but this is of rare occurrence. Marked nasal blocking should be removed. Ventilation of the sphenoid as a therapeutic measure is rarely indicated, but is still advocated when optic atrophy is imminent. The infection may be in remote regions of the body. The focus may be found in the appendix, gall bladder, prostate, fallopian tubes, genitourinary or intestinal tracts. Systemic infection following influenza, scarlatina, etc., may

be responsible for the optic nerve lesion. Local treatment is advocated in addition to the removal of the focus.

Normal vision was obtained in 63 per cent of the cases undergoing operations, while 20 per cent improved. In the remaining 17 per cent, which were chronic, further loss of vision was prevented. The ethmoids were opened but once, the sphenoid twice and the antrum four times. Over 80 per cent of the cases had infected teeth or tonsils or both.

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VIII.

A FURTHER DISCUSSION OF AFFECTIONS OF THE OPTIC NERVE DUE TO SINUS DISEASE.

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One of the notable contributions of Dr. Loeb to our specialty was his painstaking sectioning of the head to show the anatomic relationships of the optic nerve to the various accessory sinuses of the nose. He, confirming the work of Onodi, showed the most intimate relationship to exist between the optic nerve and the sphenoid and posterior ethmoid cells. The more intimate relationship sometimes varying as to whether it were with the sphenoid or a posterior ethmoid cell according to the relationship of these cells. Clinically a relationship as to disease had long been recognized as existing between the diseased accessory sinus and the optic nerve.

Dr. Loeb was led to the conclusion that the organisms or toxins from the diseased sinus were borne either arterially or lymphatically direct to the nerve, whose fibers thereby were more or less inhibited as to function, giving rise to an inflammatory condition of the nerve or optic neuritis, papillitis, etc.

I differed with him, claiming that the affection of the nerve was brought about mechanically by the disturbed circulation of the parts, and that the changes in the nerve might as easily be brought about by the disease of the sinus most distant from the nerve.

Dr. Loeb's was the theory and belief of the majority. This belief seemed to be the natural deduction from Onodi's and Loeb's work and is in many cases undoubtedly true, but should not be believed to the extent that other disturbances of the nerve from sinus disease cannot be seen. Before the works of Onodi and Loeb were published several of the older writers had associated the changes in the nerve to a thrombophlebitis of the ethmoid and ophthalmic veins. Notable among these writers were Vossins,¹ Gutmann,² Zarniko,³ Hajek,⁴ Kuhnt,⁵ Logan Turner,⁶ Denis and Vacher,⁷ Clermont,⁸ Brückner,⁹

Wurfel,¹⁰ Courtax,¹¹ Birch Hirschfield,¹² Ziem,¹³ Weiner¹⁴ and Ball,¹⁵ and this perhaps before so much attention was given to focal infection.

Dr. Leon T. White, who has written extensively on the subject of optic nerve disturbances from sinus disease, and who for some years seemed to believe in the direct poisoning of the nerve from its proximity to the foci of infection in the diseased accessory sinus, still believes in the direct poisoning, but has now arrived at the conclusion that the frequency of the nerve disturbance from sinus disease is insignificant as compared with the frequency with which it is affected from other foci, notably from diseased teeth or tonsils, and names as possible foci the prostate, fallopian tubes, etc. This may all be true as to blood borne organisms or toxins, but there is another manner of disturbance of the nerve from the diseased sinuses, not so generally recognized, which is the disturbance arising from the phlebitic ethmoid and ophthalmic veins. An almost constant pathologic finding in the mucosa of a chronically diseased sinus is a phlebitic condition of the veins, generally thrombophlebitis. These veins are contiguous to and empty into the ophthalmic veins. As the phlebitic condition advances from the ethmoid veins to the ophthalmic, the return flow of blood from all those parts supplied by the ophthalmic veins which lie distal to the anastomosis of the diseased veins will be retarded and an edematous condition of these distal parts will occur.

For purpose of comparison, let us consider the condition of the optic nerve produced by brain tumor, which all agree arises because of the retarded flow of blood due to pressure of the brain tumor on the veins. The nervehead is swollen and edematous, choked disc occurs; fibers of the nerve are separated from one another by cerebrospinal fluid. A similar condition of the nerve arises from a diseased sinus. In fact, so similar that no oculist can tell objectively whether the condition arises from a brain tumor or a diseased accessory sinus.

In either case the condition is due to an edema of the nerve brought about by a retarded return flow of blood; in the brain tumor the caliber of the vein is diminished on account of pressure on the vein; in the case of diseased sinus the caliber of the vein is diminished by the thickened wall of the vein or by thrombi.

The vein may be swollen and edematous, due to the condition of its own veins, or the swollen and edematous condi-

tion of the nerve may be brought about by pressure brought to bear on it from the swelling of the soft parts surrounding it. The pressure on the nerve might easily happen as it passes through the optic foramen or canal, due to the swollen or edematous condition of the soft parts surrounding the nerve at that point.

The accompanying pictures will show what havoc may be produced by the phlebitic condition of the veins.

No blood borne virus or organism will produce this edematous condition of the nerves.

Subjectively one of the first things complained of by the patient whose nerve has been poisoned by a blood borne toxin is blurred vision. Whereas, one whose nerve is edematous continues with practically normal vision for some time, and the condition of the optic nerve may be discovered by accident. Both conditions tend ultimately to nerve atrophy.

One who gets this condition well in mind will be able to see how it is that so frequently the disturbance of the optic nerve accompanies or arises from a so-called latent or occult sinusitis.

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Fig. 1.

Is the picture of a child suffering from diseased antra and ethmoids.

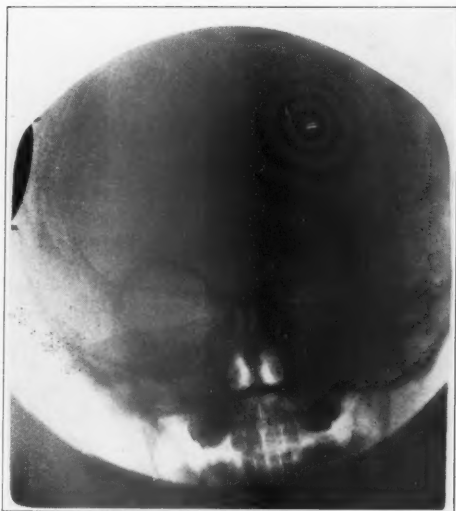


Fig. 2.

Shows the Roentgenray picture of the child's head showing involvement of all the sinuses, but the greater involvement on the left side. The child died. An autopsy was done.

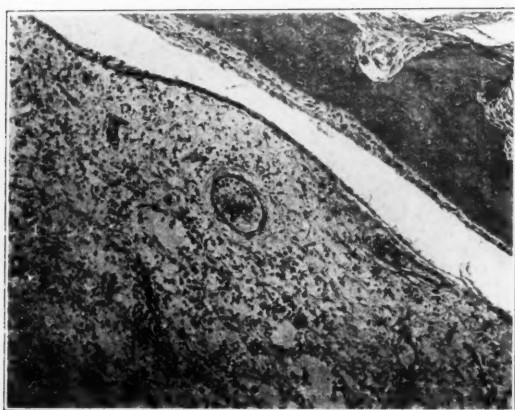


Fig. 3.

Shows a microscopic section through the ethmoidal region. The broad white streak through the picture is a section of an ethmoidal cell wall. Beneath it, in the middle of the picture, will be seen a thrombosed vein.

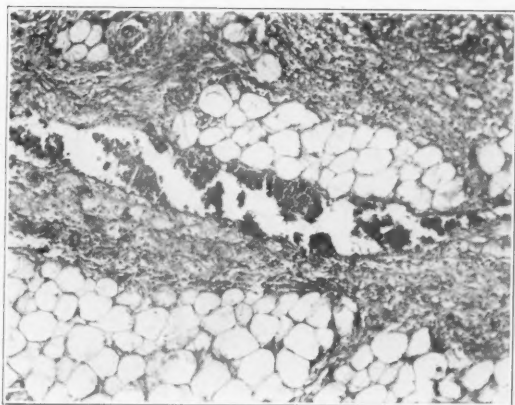


Fig. 4.

Shows a microscopic field through the orbital tissue showing thrombi in a longitudinally cut vein.

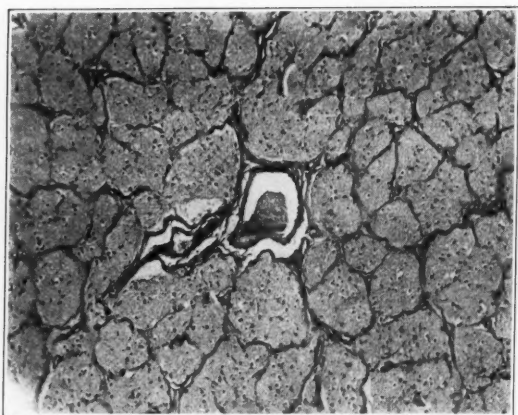


Fig. 5.

Shows an organized thrombus in the central vein. Such a pathologic condition in a living head must be accompanied by a considerable swelling and edema of all tissues whose venous drainage is via the thrombosed vein. In the particular case under discussion the drainage from the optic nerve and the orbit is into the thrombosed ophthalmic vein, the edema of the orbit producing an exophthalmus, the edema in and about the optic nerve producing the condition of nerve that I have endeavored to elucidate.

IX.

FURTHER OBSERVATIONS ON THE ETHMOID PROBLEM.*

BY ROSS HALL SKILLERN, M. D., Sc. D.,

PHILADELPHIA.

Some years ago, to be exact, in 1920, I presented my own experiences and incidentally views on this subject, thinking at the time, if indeed I gave the matter any thought at all, that it would attract but passing attention and merely be classed as the personal views of an individual observer. Basing my opinion on the unsuccessful and in many instances harmful results following extensive surgical work on the ethmoid body proper, I advocated primarily the complete removal of the middle turbinate without interfering with any portion of the ethmoid capsule. This, of course, pertained only to those cases in which no surgery had been previously attempted, or at least where the middle turbinate and underlying cells were, even though diseased, to all intents and purposes intact. Unless this obtained, the very purposes of the intent were defeated. These purposes, of course, were twofold. First, to install thorough aeration to the entire ethmoid, not only in the middle nasal passage below the insertion of the middle turbinate, but also around the superior turbinate and in the sphenoethmoid recess, through the removal of the underlying body, which acted more or less as a barrier to that region, namely, the middle turbinate. Second, to permit the infected but intact ethmoid capsule to either rid itself of the infection through aeration and drainage, or so to react against the infecting organism as to cause it to become localized in one or more cells or areas, thus making the infection amenable to logical and not haphazard surgical treatment.

Since that time, further experiences have proven that there has been more in this assertion than I had hitherto dreamed.

*Read at the annual meeting of the Pacific Coast Oto-Ophthalmological Society, at Spokane, June 7, 1927.

It is now a well known fact, as many of our sad results will attest, that even under the most favorable circumstances the ethmoid capsule reacts badly to surgical interference and, indeed, directly as to the extent. In other words, the more intentional the exenteration, the greater the likelihood of ultimate failure; and in the same manner, the less the infection or area of pathologic changes, the greater the harm results, in direct proportion to the extent of the surgical procedure. This statement may seem involved, but to use an old adage in a new light, one may say it is better to send a boy to do a man's work than a man to do a boy's, as far as the surgery of the ethmoid is concerned. In other words, it is better to do too little than too much. I appreciate that having made these statements, I am in the same position as the witness when the lawyer asked him if he still beat his wife. However, I do not propose to try to explain away the meaning of these remarks, but endeavor in the course of this presentation to show my reasons, which, if coinciding with your own experiences, may also meet with the possibility of your approval, if not actual endorsement.

Now, in the first place, I am assuming that we have one of the common garden varieties of ethmoid cases to deal with, where there is little to be seen on direct rhinoscopy outside of a juicy mucopurulent ethmoid region, with some general hypertrophy, but no signs of thick, creamy pus streaming down in a given place which constantly reappears after wiping away, as some of the textbooks would have us believe. Only during attacks of so-called acute colds in the head (which these patients complain of with great frequency and which are, of course, only acute exacerbations of their ethmoid infection) do we see any appreciable quantities of true pus such as is noted from the frontal or maxillary, and even then is mixed with or accompanied by considerably larger quantities of a translucent mucoid secretion. Little or no headache or other extraneous symptoms are present, and between the intervals of these acute flare ups the discharge from the nose, particularly that collecting in the nasopharynx during the night and manifesting itself on arising in the morning, is the one great symptom that most annoys the patient and in all likelihood the one which has brought him to you.

This, gentlemen, represents the class of cases, together with the preliminary surgery and after treatment, that I wish to bring before you this evening for your consideration. We will assume that such a one has come to us and placed himself unreservedly under our care. We will also assume, *mirabile dictu*, that no surgery has been attempted, for in the very nature of the case it is a chronic one, and it is indeed strange if he has not already passed through other hands. Straightway on examination we of the old school would immediately think of that rhinologic postulate, "Find the pus and follow it to its source." All right, try and do it. If one persists in his attempts to uncover the exact source, he will soon find that to it the proverbial needle in the haystack is as evident as a wart on the end of a nose. Pus, if any is seen at all, is either in the lower part of the nose, or, if above, is coming from everywhere in general and nowhere in particular. Even the nasopharynx gives us but little information on this point. Suction, by means of negative pressure, will oftentimes bring it out, but even after it has appeared the question arises, "Where does it come from?" Only very rarely and in the most exceptional cases, with wide noses and a small middle turbinate, could one judge with any degree of precision even the particular area from which this secretion was drawn—at least such has been my experience. Despite the fact that we have not been able to prove with any degree of accuracy from which cell or cells the inflammatory product has been secreted, nevertheless the patient under our manipulations has undergone more or less improvement and is quite sanguine of ultimately being relieved of his affliction. As time goes on, however, the true status of the condition become more and more apparent to both the physician and patient. The former sees that despite his treatments the appearance of the intranasal picture changes but little, while it gradually dawns upon the patient that at every little lapse in the continuity of the treatment he seems to catch a fresh cold, which leaves him in much the same condition as when he first came and the treatments were primarily instituted. Becoming impatient, he insistently complains of the awful discharge and nasal stoppage, which seem to grow in their intensity, treatments giving only temporary relief. This place has hitherto been the point where one, actuated by the

desire to relieve his patient in the shortest possible time, permitted his feelings to prejudice his better judgment, and with the hope that fathered the conviction, flew to surgery, oftentimes, I am sorry to say, widespread and haphazard, with the result of which all of us are, or have been, intimately familiar. As I say, once upon a time extensive surgery—that is, an attempted complete intranasal exenteration of the ethmoid—was indicated when this impasse between the ethmoid infection and the attending surgeon prevailed.

Once upon a time, yes, once upon a time red flannel underwear was all the rage, flies were swarming in every dining room and we took a bath only on Saturday night. But this has all changed, so it should be changed as far as the ethmoid is concerned; but I am getting ahead of my story. The Chinese have a maxim, "Prudence will carry a man out of his difficulties, but the impetuous will find each step the more difficult." I do not know of any better piece of advice when we have reached this place in the therapeutic handling of our ethmoid case. We have already learned that by applying surgery we have only been cohabiting with chimeras and begetting specters, for who, indeed, has ever contemplated an ethmoid some months after a so-called complete exenteration, with its conglomerate mass of hypertrophied and scarred mucosa, fibrous tissue and bone bathed in pus and carpeted with crusts that has not, indeed, seen a specter, and a very horrid one at that, particularly if he himself had performed the operation?

It will then be seen that to resort to extensive surgery at this time with the hope of a radical cure is the acme of fatuity, but something must be done, and very quickly at that, if we hope to retain the confidence of our patient. Very well, let us consider the case from the point of view of what actually confronts us and what simple measures can be applied to correct or remedy the basic etiologic factor, as the case may be. We have confronting us a unilateral infection of the ethmoid, the extent of which is undetermined, with intermittent exacerbations of discharge: sometimes a thin, watery secretion, at intervals becoming thick and purulent, then reverting again to its original thin and acrid consistency, the middle turbinate being intact. Experience has already taught us that aeration and

drainage are as essential to the ethmoid mucosa, in order to rid itself of infection, as oxygen is to the lungs, and of the two aeration is far the more important. If we have good aeration the drainage will take care of itself. Well, what prevents the inspired air from freely circulating around, over and through the cells of this diseased and inflamed labyrinth? First, the body of the middle turbinate, and second, the swollen and oft-times polypoid mucosa. Very well, what are then the indications to bring around this desired aeration? Naturally, to remove the obstruction thereto, namely, the middle turbinate. Let me say here that this is the point where I disagree with some of my friends, who consider this structure of great physiologic moment in nasal respiration and who make every effort to preserve it intact by performing an excavation of the cells between it and the orbital plate. I cannot agree with them in this respect, nor can I subscribe to the importance of all the functions which they attribute to it. It seems to me, if one accepts that aeration is the *primum desideratum* in the ultimate cure, that to keep the middle turbinate in place defeats the very object of the intent, for this body acts as a barrier in directing the inspired air with its high oxygen content from and away from the posterior ethmoid regions. The middle turbinate is then removed in its entirety by the method most favored by the individual operator. Personally I prefer the scissors and snare, removing as much as possible with the first constriction of the wire and leaving permanently the posterior end to about the size of a small pea. I have found by leaving this portion that hemorrhage, both immediate and post-operative, has become a thing of the past. This is due to the fact that the large nasopalatine artery branches lie just behind this point, so that by avoiding the extreme posterior end of the turbinate injury to this artery is also avoided. The removal is made on a line with its insertion into the ethmoid body proper, thus keeping well below the terminal fibrils of the olfactory nerves and avoiding the possibilities of the dangers incurred in opening their perineural sheaths, which, of course, lead directly into the subarachnoid space.

As soon as this turbinate is removed, our operative technic should be suspended, except perhaps for the removal of ragged portions of bone or mucosa which happen to remain. The main

point is to leave the body of the ethmoid absolutely intact, as much as it pains us, for who has not seen the polypoid changes and even small polypi revealed around the neighborhood of the bulla which seem to so hospitably invite their removal as soon as the overlapping turbinate is out of the way? Our enthusiasm in this regard may be the very cause of our undoing, as I have found that to continue at this stage is to distinctly overdo it, for in endeavoring to remove this redundancy of hyperplastic tissue we not only take it, but also a certain amount of healthy tissue in the immediate vicinity, both of which would have in all likelihood returned to normal following the aeration made possible through the removal of the turbinate. So that our one endeavor is to completely and cleanly resect this structure and discontinue any further surgical manipulations, at least for the time being.

Now, just as in a gastroenterostomy for duodenal ulcer, here also this operation should be considered as a preliminary procedure to subsequent treatment, for without one the other loses most of its virtue. In four or five days, or as soon as healing has well set in, the local treatments should be instituted under the following hypothesis: First, that the extent of the infection through the ethmoid body is unknown, and second, though marked hyperplasias be present, even to actual polyp formation, the area of subsequent tissue resolution to normal through the newly created aeration and under appropriate forms of treatment is also unknown. Our first endeavors should be directed towards ascertaining, as far as humanly possible, the extent of the infection through the ethmoid labyrinth, after which treatment can be applied that may be termed fairly scientific and does not favor the haphazard methods which consisted of all that we have hitherto had under our control.

About five days, then, after the removal of the turbinate, on inspection, the whole ethmoid capsule on that side has undergone a marked change. Instead of a mass of acutely infected, swollen and hypertrophied tissue, the entire picture has changed, giving one the impression that the inflammation has not only markedly lessened, but also shows a tendency to congregate in certain places, with areas of mucosa intervening

which apparently have entirely regenerated, especially if marked hyperplasia had been uncovered when the turbinate was first removed. Even small solitary polypi have reduced themselves almost to disappearance. Now is the proper moment to use our method for the purpose of ascertaining the spread of the infection. This can be done by applying some substance which will not only tend to draw the inflammatory products out of the infected tissues, but also to act as a germicide as well as have a beneficial, curative action on the diseased mucosa. This I appreciate is a large order, but I feel convinced we have found a true panacea in tampons about the size of one's thumb saturated with a strong solution of colloidal silver. Many firms manufacture such a drug, which goes under the name of argyrol, silvol, cargentos, silloid, etc.; all are claimed to be nonirritating, so that I shall play no favorite. There are, however, several conditions in their application which must be observed: First, the solution must be strong (25 to 50 per cent); second, the solution must be fresh; third, the solution should be hot or at least warm; fourth, the tampons must remain in place from four to six hours. These tampons while in place act in a similar manner to a hot poultice, but in a two-fold manner. First, they tend to draw the inflammation toward the surface, and second, have a decided bactericidal action on the infecting microorganism as well as possessing a healing quality beneficial to mucous surfaces.

Briefly, then, the entire method is carried out as follows: Both nares are thoroughly cleansed with warm normal saline solution. A suitable piece of cotton is fashioned into a tampon about the size of one's thumb and partially soaked in a fresh, warm, strong solution of the colloid silver; then gently pressed into the upper portion of the nasal chamber until it covers the entire ethmoid region. The tampon, as I said, is permitted to remain in place from four to six hours. These treatments are carried out every other day until the inflammatory symptoms have greatly subsided, then gradually tapering off until they cease entirely. The length of time varies with the individual case, but, roughly speaking, one can count on from four to six weeks for the average case.

These cases that we have just described, it must be remembered, were those in which no surgery had hitherto been at-

tempted. Now let us consider that group in which multiple operations had been performed and the whole ethmoid region represented a thoroughly disorganized and semidense suppurating mass with all landmarks, as far as the ethmoid itself is concerned, more or less obliterated. Further surgery merely adds fuel to the fire, as the resulting granulation tissue soon becomes reinfected, making the condition worse with each succeeding operation. If such a case comes under our care, we must take it up from a systematic point of view and first rule out suppuration from the true sinuses. In the presumption that this has been accomplished, we now endeavor to ascertain from which portion of the ethmoid the inflammatory exudate is secreted. Sometimes this is made possible by using the tampons and on removing them noting which portion is covered with pus or greatly decolored. If the diseased portions can be brought to light by this procedure, appropriate treatments to those localities can be immediately instituted. Suppose, on the other hand, the precise source of the purulent discharge remains undisclosed, the pus appearing to be everywhere, yet coming from nowhere. These cases require the most thorough and farreaching surgery that we are ever required to practice intranasally with reference to the ethmoid. I feel, and have felt for some time, that to reach all the cells that are accessible to intranasal manipulations, it is essential to remove the uncinat process from its insertion to its end. I am also quite aware that even with this structure out of the way the entire ethmoid labyrinth cannot be removed endonasally, but as a matter of fact this is almost never indicated, as all the cells are not always affected, at least to the same extent as to require complete exenteration. In any event, after the uncinat is out of the way, from one-quarter to three-eighths inch more room is gained, which may give us just the amount of space necessary to successfully carry out our applications. The hot tampons are continued here just as before, although the occasional removal of polypoid or excessive granulation tissue may from time to time be found necessary. Improvement, however, will surely come, and if the final results do not measure up to 100 per cent, nevertheless, if we have relieved the patient to such an extent that he says, "Why, Doctor, the little inconvenience that I now have is nothing to what it was before the treat-

ments," we should be content with our efforts, for no one can expect an ethmoid which has been torn to tatters by infection and instrumentation to ever functionate as a normal one and not be a place of minor resistance for every cold to which the patient is subjected.

And in conclusion, gentlemen, let me state that I firmly believe, as time goes on, we will no more look upon our treatments in the light of a hope rather than a faith, but will finally agree upon a standard method of handling these cases, with such uniformly happy results that the subject will no longer be termed "The Ethmoid Problem."

X.

STUDIES OF PATHOLOGIC TISSUE REMOVED
FROM CHRONICALLY INFECTED NASAL
AND ACCESSORY SINUSES: A PRE-
LIMINARY REPORT.

By W. V. MULLIN, M. D., AND R. P. BALL, M. D.,

CLEVELAND CLINIC.

CLEVELAND.

GENERAL DISCUSSION BY W. V. MULLIN.

For some years I have been trying to correlate the infections within the nose and the nasal accessory sinuses with the symptoms of these infections—whether local or general. The pathways of the lymphatic drainage from the nasal sinuses have been of considerable practical value to me, but while bacteriologic studies in many instances showed the same organisms in the discharge from the bronchi as were found in the infected sinus or sinuses, still these threw no particular light on the relation of the symptoms to the pathologic conditions produced by the infection. In various cases, I have had the tissues which were removed from the sinuses and nose at operation examined pathologically. The reports of these examinations were accurate; they told me just what the tissue was and what it contained histologically, but they still gave no pertinent information. I gave up the problem for a time, but during the past year took it up again with the same fruitless results. I was much discouraged; but still I could not be reconciled to the fact that the patients upon whose sinuses I operated for apparently identical conditions showed such a variety of symptoms. Surely, the pathology in these cases must differ in some way. I was particularly troubled by the observation that while in some cases of polyposis the patients would have only nasal symptoms, in others chronic bronchitis would be present, and in still others they would have bronchial asthma. In desperation, I decided to analyze my cases backwards, as it were. I therefore selected a number of what might be called terminal

cases—that is, long standing cases of pronounced chronic sinus disease in adult patients who presented a variety of symptoms. I asked Dr. Ball to make a careful microscopic study of these cases in order to see if he could arrive at some histologic and pathologic differentiation. He will give the results of his investigation.

The following classification is presented with the sole purpose of providing a working basis for the desired correlation:

- I. Local symptoms.
 1. Pain.
 2. Discharge and odor.
 3. Nasal obstruction.
 4. Bone changes, osteomyelitis.
- II. General symptoms.
 1. Cough.
 2. Bronchitis and bronchiectasis.
 3. Bronchial asthma.
 4. Arthritis.

In these studies, no attempt has been made to discuss the relationship between nasal diseases and bronchial asthma. No distinction has been made between patients with bronchial asthma who were allergic and those who were not. The only point considered in this study is that all of these patients did have chronic infected sinuses that called for operative treatment and that certain of these patients had bronchial asthma.

I have found very little connection between chronic infected sinuses and chronic arthritis, therefore our study of cases of that type has been decidedly limited.

PATHOLOGIC STUDIES BY R. P. BALL.

The lining of the nasal cavity, particularly of the "respiratory portion," is very vascular. The outer portion of the cavity is composed of an epithelial layer consisting of pseudostratified columnar, ciliated epithelium, while the underlying tissue is made up of a fibrillarlike structure which contains numerous thin walled vascular channels and tubular glands of the serous and the mucous type. The lining of this portion of the nasal cavity is thicker than that of any other portion. The accessory nasal sinuses, the maxillary sinuses in particular, have a similar but much thinner lining which contains fewer glands. The epithelial lining of the sinuses is often of the thickness of a

single cell or, at the most, of only two or three cell layers. The cilia have not always been seen in the tissues examined. For convenience, with the exception of neoplasms, we have classified the histologic changes in four groups, according to the presence of the following conditions: (1) edema, (2) fibrosis, (3) leucocytic reaction, and (4) glandular hyperplasia or atrophy.

Edema.—To use the term edema in this connection may seem somewhat farfetched, since there is always a certain amount of serous exudate in any type of inflammatory reaction. Polypi, however, have been removed, which have been relatively free from leucocytic reaction and in which no histologic change was seen excepting wide connective tissue spaces filled with serum. Grossly these polypi are recognized as semi-translucent, ovoid, smooth, moist, soft and usually pedunculated tumors which have a moist homogeneous cut surface, from which drips a clear serous fluid. This description is applicable only to the purely edematous polyp. The surface may be broken and covered with a fibrinous exudate, while, because of inflammatory changes, the cut surface may present a mottled grayish or darkened hemorrhagic appearance. It is not probable that tissue curetted from the bony wall of a sinus or nose will be mistaken for edema of the normal "erectile" tissue, since in the former the edematous fibrillar stroma shows wide interstices filled with serum rather than an engorgement of the vascular channels.

Fibrosis.—Although fibrosis in varying amounts is sometimes found, it is not always associated with a leucocytic reaction. Its presence suggests a healed fibrosed state. Fibrosis is most frequently found in cases which show a leucocytic reaction in masses which produce minute abscesslike cavities.

Leucocytic Reaction.—In every case examined the tissue removed from the maxillary sinuses showed leucocytic reaction in varying quantities and types. The mononuclear leucocyte is the type of reacting leucocyte most frequently encountered, while the greatest number of cells are the lymphocytes and plasma cells. Although eosinophiles are practically always present in cases of asthma, they are not confined always to asthmatic cases, in all of which, however, eosinophiles were found in abundance—sometimes being almost the only react-

ing cell encountered. In all cases, polymorphonuclears are usually present, but not in the great numbers which are present in lesions with pus formation which occur elsewhere in the body.

Glandular Hyperplasia and Atrophy.—Except for distension with mucoid material, changes in the glands are seldom found. Sometimes in the maxillary sinuses the distension is so great that it produces the appearance of a large thin walled, semi-transparent multilocular cyst, which collapses when an attempt is made to remove it. Microscopically these "cysts" appear as large cavities lined with a single layer of cuboidal and columnar epithelium and filled with a neutrophilic or acid staining structureless substance. No localized group of hyperplastic glands which suggested an adenoma or papilloma has been encountered. We believe this to be of great significance as indicating the type of treatment to be used in cases of recurring polypi.

In Table I are given the findings in 32 selected cases. At present no absolute constant is demonstrable which would indicate whether the patient had "local" or "general" symptoms. In the asthmatic cases the histologic findings and the symptoms are the most closely related, but even among these the findings are not constant. The tissue of the large edematous polypus has such a small number of fibroblasts and the interstices are so large that this type of polyp cannot be considered as a fibroma molle. If the serum is pressed out or the specimen is dehydrated in alcohol its thickness diminishes to that of the lining of the nasal cavity from which it originated. If this polypus were an edematous fibroma we believe it would show fibroblastic proliferation such as is seen elsewhere. Edematous fibromata are found occasionally, but usually in the region of the posterior pharynx and larynx rather than in the nose or accessory nasal sinuses.

COMMENT.

This group of cases was selected from over a hundred specimens which have been examined. When the examination was made the pathologist knew nothing of the clinical symptoms. The sections were prepared from paraffin embedded tissue and were stained with hematoxylin and eosin.

TABLE I.
HISTOLOGICAL FINDINGS IN A GROUP OF THIRTY-TWO SELECTED CASES OF DISEASE OF
THE NASAL AND ACCESSORY SINUSES.

No.	Clinical Symptoms or Diagnosis	HISTORICAL FINDINGS						Symptoms
		Edema	Fibrosis	Polys.	Eosino- philes	Mono- nuclears	Glandular hyperplasia	
1	Nasal polypi, recurrent nasal ob- struction.	++++	0	0	++	+	0	Local
2	Nasal polypi, recurrent nasal ob- struction.	+++	0	+	++	++	0	Local
3	Nasal polypi, recurrent nasal ob- struction.	++	+	+	++	+	0	Local
4	Pain, foul discharge.	+	0	++	+	+	++	Local
5	Nasal polypi, recurrent (?) nasal obstruction.	+++	0	0	+	+	0	Local
6	Pain in maxillary sinus associated with abscessed teeth.	+	++	++	+	+	+	Local
7	Pain in maxillary sinus associated with abscessed teeth.	+	0	+++	+	+	0	Local
8	Nasal obstruction.	+++	0	+	+++	++	0	Local
9	Nasal discharge with foul odor.	+	+++	++	+	++	0	Local
10	Nasal discharge, chronic cough.	0	++	0	0	0	++	Local and general
11	Pain (Osteomyelitis).	+	+	+++	+	+	+	Local
12	Pain, septicemia (Osteomyelitis).	+	+	+++	0	+	0	Local and general
13	Cough (Bronchitis).	0	++	0	0	0	+	General
14	Pan-sinusitis (Bronchiectasis).	+	0	++	+	+	0	General

15	Cough, sputum (Chronic bronchitis).	O	+	+	++	+	O	+	General
16	Chronic bronchitis.	+	O	+	++	++	O	+	General
17	Chronic bronchitis.	++	+	+	+	++	O	+	General
18	Slight cough.	+++	O	+	+	++	O	+	General
19	Bilateral bronchiectasis.	O	+	+	+	++	+	+	General
20	Chronic cough.	++	+	O	O	+	+	+	General
21	Chronic cough.	+	O	+	O	+	O	+	General
22	Multiple arthritis.	O	++	+	++	+	O	+	General
23	Multiple arthritis.	O	+	++	+	++	O	+	General
24	Bronchial asthma.	+	O	O	+	+	+	+	General
25	Bronchial asthma (?).	O	+	++	+	+	O	+	General—patient later died with cerebral hemorrhage. Autopsy was done. Lungs and bronchi normal.
26	Asthma.	++	O	O	++	+	O	+	General
27	Asthma.	++	O	O	++	+	O	+	General
28	Asthma.	+	+	+	++	+	O	+	General
29	Asthma.	+	O	+	++	+	O	+	General
30	Atypical asthma.	++	O	O	++	+	O	+	General
31	Asthma and tuberculosis.	+++	O	O	++	+	O	+	General
32	Asthma.	O	++	O	++	++	+	+	General

O—None. +—Occasional. ++—Slight. +++—Moderate. ++++—Marked. +++++—Almost entirely.

There have been no positive findings, which excludes any general group. However, some types are distinct and further studies are indicated to give more information on this interesting and important group of cases.

SUMMARY.

A preliminary report of the histologic changes found in a selected group of cases of diseases of the nasal and accessory nasal sinuses is presented.

No constant finding is present which will enable one to correlate a definite group of symptoms with the pathologic findings.

Certain changes are sufficiently constant, however, for their absence to permit a negation of the presence of a disease focus in the sinuses.

The most severe type of sinus infection, as judged by the destruction of tissue and the polymorphonuclear leucocytic reaction, is found in cases of bronchitis and of cough without local pain.



Fig. 1. Photomicrograph of a section of tissue from a maxillary sinus showing a small abscess surrounded by edematous tissue and early fibrosis (x160).



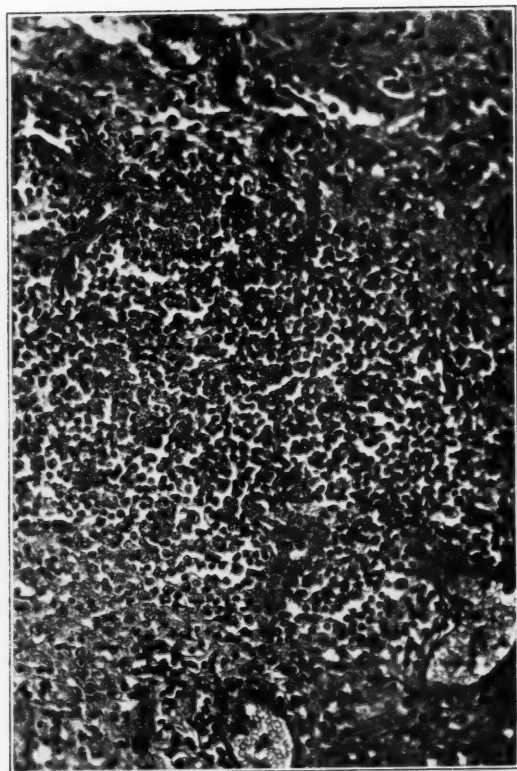


Fig. 2. A higher magnification (x250) of the section shown in Fig. 1.



Fig. 3. Photomicrograph of a section of the nasal mucous membrane showing a large number of serous and mucous glands with a clean stroma (x160).





Fig. 4. Photomicrograph of a section of tissue from a maxillary sinus showing distended mucous glands lined with tall columnar epithelium and filled with mucoid like material.

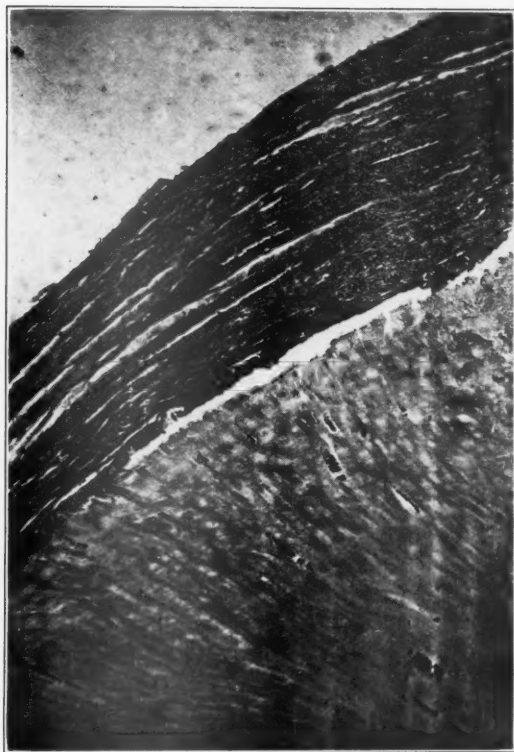


Fig. 5. Photomicrograph of a section of tissue from a maxillary sinus showing a markedly distended mucous gland. The lining cells are compressed and of low columnar type; compare with Fig. 4 (x160).



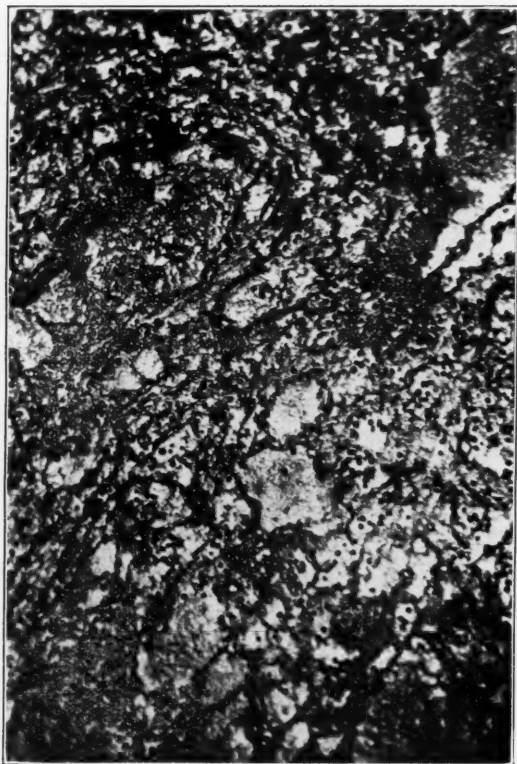


Fig. 6. Photomicrograph of section of a nasal polyp showing marked edema. The interstices are large and filled with serum (x160).

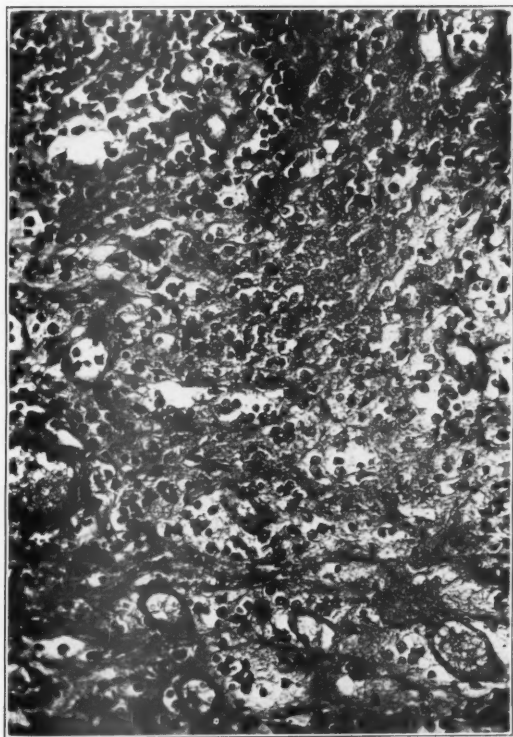


Fig. 7. Photomicrograph of a section of a nasal polyp showing edema and mononuclear leucocytic reaction. The blood vessels are distinct and are not confused with interstices (x250).



XI.

THE WHY AND THE HOW OF HARELIP CORRECTION.

BY VILRAY P. BLAIR, M. D.,

ST. LOUIS.

Nothing short of one's best could appropriately be offered in connection with any testimonial or memorial effort to Dr. H. W. Loeb who, aside from and beyond his purely surgical attainments, did so much to raise the general standard of medical education in this city. Appreciating the great honor of contributing to this volume, I carefully considered what of the little I have accomplished might in a small way bear some relation to the quality of the great accomplishments of Dr. Loeb.

Nearest to my heart is that lowly subject, the treatment of harelip, which, judged by a comparison of the theoretical possibilities with the average result, one might think to be the most difficult of all surgical operations. I have little new

to offer and use methods that were standard in the first half of the last century. Not only have I yet to show a single case disproving Bilroth's statement that there never had been a perfect operative result, but I still not infrequently have results of my own that leave more than a little room for improvement. All that I can claim after twenty-five years of intensive study is, I think, a little deeper insight into the problem,



possibly a little better selection and adaptation of methods, as evidenced by a very slow but a continuous improvement in our results, and a still hopeful attitude.

This imperfect showing is here presented, not to parade results, but to emphasize failure and need—not absolute failure, but failure relative to the possibilities—not a need for new methods, but for a deeper appreciation of sound underlying principles, a greater skill in applying them, and the avoidance of false moves.

The simple closure of the open lip is life-saving because it helps control respiratory infections. A good repair makes not only for better health and happiness throughout life, but materially increases potential usefulness. By a good repair I mean a pleasing appearance with free breathing space that will be followed by approximately normal dentition and occlusion and almost symmetrical contour. I know of no plan that will give one hundred per cent good results, but some are better than others. Some plans are too simple to be highly effective, some lack continuity, some are unproductively complicated, and there are still others with inherent vicious possibilities that will lead to their ultimate discard.

Among the basic so-called improvements in method which have been offered in the past half century, the early closure of the lip will, I believe, stand the test of time, even if it accomplished nothing more than to permit a mother to exhibit her two-weeks-old baby for which she need offer no apology. However, there are other and more permanent advantages of this practice which was popularized by Wolff, Brophy and Lane.



THE NORMAL LIP:

A casual examination of the average upper lip in the newborn shows a nose with a tendency to "turn up," symmetrical nostrils sharply bounded below, a lip relatively short from above downward in the midline, and with the mid-point of the vermillion border standing well forward of the chin and usually of the lower lip. There

SHOWING FORWARD POSITIONS



is a medial vertical groove running from the columella to the vermillion border, bounded on either side by a slight ridge. These are emphasized because in the harelip all of them are more or less distorted, and any corrective operation that fails to re-establish them will be proportionately displeasing, and subsequent growth will be more than apt to produce increasing distortion. However, I do not consider it surgically practical to restore the filtrum, at least one lateral ridge of which is always missing, but if the gross contour of the lip is good this defect is not very noticeable.

It will simplify matters to consider only "single" harelip, which term for practical purposes will here define that part of a single facial cleft that is anterior to the incisor foramen.

THE CLEFT LIP:

OF AN OPERATED AND OF A

nose, or a slight notch in the vermillion border with a fault extending up to a widened nostril, or a separation limited to the lip, floor of the nostril and alveolar process. These are differences of degree rather than of kind, and the treatment

This may be part of a continuous open cleft running through the uvula and the floor of the

of each is basically the same. In all stages there is some actual lack of tissue at the lower border of the lip, some lateral displacement of the two halves of the lip to either side of the cleft, and some flattening of the nostril on the affected side, the degree of each being in some proportion to the width of the cleft. The specific objectives of the operation should be to bring about the best possible immediate restoration of the lip, ala and floor of the nostril. If these are well accomplished subsequent growth should further improve the result by bringing the external nose to the midline and gradually closing the open alveolus, thus giving an approximately normal relation to the dental arches and producing a palate condition that can later be closed by midline suture of the mobilized soft tissues. Any sort of lip closure, no matter how unsightly, will usually be followed by a good approximation between the maxilla and premaxilla, but if the floor of the nostril is made too narrow this produces permanent obstruction; if too wide, it will be followed by increasing displacement of the external nose to the opposite side.

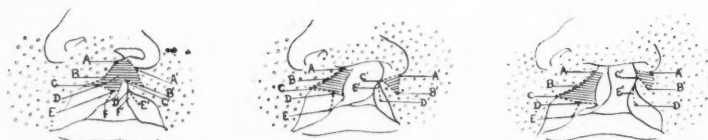
TYPES OF OPERATION:

Harelip operations date from the time of Celsus. He and others following him freshened the borders of the cleft, mobilized the lip and cheek by undercutting, and approximated with simple suture. Later the fine points of the art were lost, and even in the eighteenth century escharotics, clamps and bandages were still substituted for the rational treatment of the ancients. It is during the second quarter of the nineteenth century that we find the earlier practices again recommended.

NORMAL UPPER LIP IN THE



INFANT RELATIVE TO THE CHIN



SEVERAL COMMON DEGREES OF SINGLE LIP CLEFT ARE SHOWN ABOVE

Straight denudation of the cleft borders, which is still extensively used, gives symmetry but tends to make a lip that is long from above downward, at the expense of its lateral width, and with some loss of the desirable prominence of the central part of the vermillion border. The first recorded attempt to improve upon results obtained by the simple straight incisions was published by Husson in 1836. He used the concave incisions, and others have since substituted angular incisions. These latter, together with the Nelaton plan, form a general type of operation in which the incisions on each side of the cleft are still rather symmetrical but which tend to reproduce the natural fullness and prominence of the vermillion by forcing tissue from the cleft borders downward into the lip border.

In another classic type of operation for single lip cleft a vermillion bordered flap is taken from one side of the cleft and turned downward to piece out the vermillion along the lower border of the lip. Of the operations of the latter type I believe the Mirault, published in 1844, is, when skillfully performed, capable of giving a more perfect imitation of the normal pyramidal form of the baby's lip, and this I adopted ten years ago when I discarded the Husson as a routine procedure. The Mirault operation is, however, more difficult to handle than the Husson, Rose or Nelaton, and this fact in itself is a strong recommendation for the simpler type of operation.

AND BELOW ARE THE IMMEDIATE RESULTS OF THE OPERATION IN EACH





CASE. IN PROPORTION TO THE EXTENT OF THE LIP CLEFT THERE IS

Of these three simpler operations I think the concave incision of the original Husson is the most plastic. No matter which type of operation is chosen, the individual results will be better and skill will be more quickly acquired if one sticks to a single kind and also develops the habit of accurately measuring and marking before vision is obscured by blood.

RECORDS:

There is nothing that will help one to correct his faults and improve the good points of his operative technic like accurate readable visual records. There are three things that influence a result: first, the quality of operation; second, subsequent scar distortion; third, subsequent growth. As a rule, scar contraction of good noninfected scars is negligible, as it usually lets go in time, and within a year the condition is a replica of what it was immediately after operation. On the other hand, the variety of results that may occur in the earlier part of one's practice can be most bewildering. If one has photographs, drawings and casts of what was done at each operation, and of the condition at the various stages, it will take very much less time and fewer failures to sort out the desirable from the undesirable procedures than if one undertakes to judge from written descriptions and memory. For instance, the forceful approximation and wiring of the maxilla gives an immediate condition

LATERAL DISPLACEMENT OF THE COLUMELLA AND DISTORTION OF THE





ALA. THE EFFORT WAS MADE TO REPLACE THE COLUMELLA, RESHAPE

that very much facilitates the closure of the previously wide spread lip cleft, but our records of these cases during their subsequent growth showed that in a few distressing growth distortions occurred that caused us to abandon this type of operation. The five last cases shown on this and the next page were repaired over a wide alveolar cleft. Subsequent spontaneous closure of the bony cleft will largely correct the obliquity of the mouth.

PRELIMINARY MARKING:

The marking points are made by pricking with a hypodermic needle or "crow quill" pen dipped in an aqueous solution of methylen blue and are so placed as not to be eliminated by the cuts; in this way they can also serve as guides in placing the key sutures.

PLANNING:

To have results approach the ideal, extreme accuracy is necessary in planning, cutting and suturing; a sixty-fourth of an inch one way or the other will make a noticeable difference. I believe it was my late and revered friend, Dr. James Thompson, who introduced the use of the compasses or dividers into lip surgery, a practice that cannot be too strongly recommended, but in the final analysis it is the eye that will give the most accurate check. If the denudation incisions are placed

THE ALA AND TO CLOSE THE CLEFT WITHOUT UNNATURAL LENGTHENING





OF THE LIP AND AT THE SAME TIME PRESERVE THE FULLNESS AND

too far from the cleft borders, then the sutured lip will be distorted: if not far enough, it will leave an ugly lateral prominence to the finished lip and most likely a notch. Of the two faults the removal of too much tissue is the greater sin, for one can always go back and remove more.

THE NOSTRIL:

Whether the cleft is wide open or just a notch in the vermillion border, there will be some spreading of the nostril. If this deformed nostril is carefully examined it will be seen that in most cases it is flattened from before backward in some proportion to the extent of the lateral spread, that the long axis of the opening is rotated away from the vertical towards the transverse. Better results, both cosmetic and functional, will follow a correction that in part rotates the axis of the nostril rather than relying entirely upon removing tissue from the floor.

In infants or young children this rotation can usually be partially or completely accomplished by mobilizing the base of the columella and related nasal mucosa, contouring of the ala, and proper suturing of the floor.

In older children, over three or five years, this simple mobilizing of the columella and septal mucosa may not be sufficient. In such cases the columella is deeply split longitudinally.

PROMINENCE OF THE LIP AND THE VERMILION BORDER.





ADJUSTMENT FOR

ward, which will allow the displaced nostril to be rotated into its proper position. Replacing the nostril also restores the external contour. Normally the extreme lower end of the ala curves around until it points rather directly toward the base of the columella. In the wide open cleft it may point almost straight outward. With various amounts of spreading, any de-

terminally and the half columella on the cleft side, together with the adjoining mucosa, thoroughly freed by deep undermining, preferably without breaking through the septal mucosa. Next the skin of the tip of the nose is undermined and a triangular piece of the latter is removed from above the displaced nostril. This permits the freed half of the columella to be sutured for-



OLDER CHILDREN.



gree of lack of incurving may be found. In correcting the spread nostril it is essential that the ala be rotated to conform to its fellow. In doing this to an ala that is flat the lining skin will buckle in a way to obstruct the lumen. In order to overcome this and the accompanying droop of the free border of the ala, an incision is made in the lining of the outer wall and roof from

the floor to the septum approximately along the upper border of the lower lateral cartilage, and below this cut the skin is freed from the underlying cartilage as far as the free border of the ala. This freeing of the bulging lining permits the apparent surplus to be drawn up into the ventricle and into the outermost part of the vestibule where it belongs, the new relation being maintained by through and through mattress sutures.

It takes all of the above and more to give the best that is in harelip surgery. Today the majority of people are really seriously concerned about their appearance, and facial surgery has progressed to the point where to be acceptable it must be at least relatively pleasing; the mere surgical closure of the lip cleft can of itself no longer be considered adequate.

From the Department of Surgery, Washington University School of Medicine.

XII.

MEDICINE IN RETROSPECT.

By CHARLES H. MAYO, M. D.,

ROCHESTER, MINN.

It is a privilege to contribute to this memorial number in honor of the accomplishments of Doctor Hanau Loeb. How fortunate we have been who have lived during this great period of medical progress of say, thirty years, yet I have heard my father say the same, many years ago. The textbooks of the days of his youth, when the microscope was revealing only the most obvious things and before the germ theory of disease was put forward, show in a most startling way how false their ideas of disease were. Their clinical observations were often strikingly accurate, but sometimes unaccountably faulty. It took at least a generation for Virchow's discoveries to permeate medical teaching. Yet they called themselves modern and looked back at their predecessors with a patronizing sort of pity from the pinnacles of scientific success to which they had attained. In spite of the soundness of our present theories and the exacting proof to which all our beliefs are put, I cannot always be sure that a century from now somebody will not be saying the same sort of things about us. It may be that we are sojourning in the wilderness, that such discipline is necessary for the children of Medicine, and that we have not yet so much as seen the promised land. Perhaps somebody may run across these lines in fifty or a hundred years and comment with tolerant pity: "Well, he had a glimmering of vision, anyway!"

This may all be true, but it is just as well we cannot believe it. If we are doing our "forty years," it is fortunate we do not know it. I believe we are traveling the road to the elucidation of all disease, but the journey's end may be a long way off. Each day is a step, and there is always a milestone just around the corner; that is good enough.

Beginning in 1888, I had considerable experience in diseases of the nose, throat and larynx, and visited many clinics to watch the diagnostic and operative work of famous men who did this work in a most skillful manner and discussed it in a most intelligent and interesting way. The problems in these specialized fields have grown since those days, and now they require a greater knowledge of the whole body. Such diseases do not, and, in the very nature of disease, cannot stand alone. No matter how apparently local its manifestation may be, it is becoming clearer with each new advance in medicine that disease tends to permeate the entire body. The cause is often to be sought in the perversion of some generalized function or state, such as metabolism, the acid-base balance of the blood or tissues, or that vague thing we call resistance. The symptoms are rarely localized; even the prick of a pin calls forth quite a diffuse physiologic response. And it is more and more evident that treatment can be no more properly considered local in its scope than can cause or symptoms. When a healthy man breaks his leg, we usually find that local treatment suffices; but now and then difficulties and disappointments remind us that such injuries are far from local in their effects and that the demands which local injuries make on remote functions cannot always be ignored. On the other hand, there are a score or more of diseases of other organs which manifest themselves at some time during their course by certain abnormalities in the mouth. Such evidence may be negligible from the point of view of the laryngologist, but may be of the utmost importance to the patient.

Thus the specialist's practice exacts wider knowledge of the body in health and disease; he must be able to examine the entire body if he is to understand the local lesion which confronts him and treat it as intelligently as the ever widening scope of medical knowledge permits. At the same time the increasing intricacy of his own special field tends to make him subdivide his specialty, or, as somebody has put it, to "know more and more about less and less."

Those who have grown with their specialties have adapted themselves to these two contrary demands. The young man entering such a practice may keep himself abreast of his oppor-

tunities either by apprenticing himself to a specialist of broad experience or by entering group practice. Once he has become a component part of such an organization, he must be able to assess his cases completely. His diagnosis must not be a patchwork but a woven fabric.

XIII.

HEMORRHAGE FROM THE THROAT (HEMOPTYSIS NOT OF PULMONARY ORIGIN).

BY SIR ST. CLAIR THOMSON, M. D.,

LONDON, ENGLAND.

The laryngologist is not infrequently asked if he can find in the throat the source of origin of expectorated blood, but, as a matter of fact, hemorrhage from the throat is rare, and is, as a rule, only secondary to some serious local affection. Otherwise, the blood, with few exceptions, comes from the lungs. This source is often overlooked, as the misconception is general that blood from the lungs must be coughed up, or be frothy from admixture of mucus, or be accompanied by physical signs in the chest. But in the early stages of pulmonary tuberculosis there is no catarrh or mucus to become mixed with the blood, and no cough, so that pure blood from the lungs may cause no symptoms until it is simply hawked or cleared out of the pharynx.

Indeed, blood of pulmonary origin often appears to simply well up into the mouth. The patient may wake in the night and find his mouth filling with blood; or, with one easy cough he is able to spit out a teaspoonful, or a teacupful, of bright blood, almost pure or with a little mucus, and not necessarily frothy or mixed with sputum.

To distinguish between pulmonary and buccopharyngeal blood is sometimes difficult.

L. de Reynier, a Swiss laryngologist with much experience in this subject gained at Leysin, makes the following suggestion:¹ If we carefully inspect the expectorated blood it is generally bright red at first, becoming purple or dark brown in the course of the following hours or days. Now the blood stained material from the pharynx—according to de Reynier—is only expectorated in the morning hours and may reappear every morning for weeks or months. He is of opinion that a good test is to throw the sputum into water; if the blood comes

from the pharynx, larynx or gums, it is at once dissolved on shaking it in the water; if the blood comes from the lungs, it remains in one mass and insoluble.

Hemoptysis from the lungs may be profuse and yet neither auscultation, percussion nor X-rays may be able to detect any physical sign of its source or origin.

Hemoptysis may also occur in nearly every disease of the lungs and air passages, including such affections as emphysema, influenzal bronchitis, dilated bronchi, infection of the lungs by streptococci or the bacillus coli, syphilis of the lungs, localized pneumothorax, leakage from an aneurism, bronchial fluke (*Paragonimus Westermanii*), mitral disease, atheroma and high blood pressure.²

The other sources of hemoptysis can be tabulated as follows:

1. Epistaxis, when the blood flows backwards.
2. Adenoids.
3. Enlarged veins in the pharynx and around the base of the tongue, especially in gout, cirrhosis of the liver and influenza.
4. Suppuration and ulceration in connection with malignant disease, syphilis, peritonsillar abscess and (rarely) lupus or tuberculosis.
5. Spongy gums.
6. Multiple telangiectases.
7. Vicarious menstruation.
8. Trauma from accidental injury, the passage of instruments, rupture of veins by vomiting, or surgical operations.
9. Laryngeal hemorrhage, especially in the acute laryngitis of influenza.
10. From the trachea—varicose veins, congestion from pressure of enlarged thyroid gland or of an aneurism.³
11. Various blood conditions—purpura, scurvy, pernicious anemia, leukemia, hemophilia, mercurial stomatitis, phosphorus poisoning, cirrhosis of the kidneys or liver, and certain acute fevers, especially enteric and yellow fever, hemorrhagic small-pox and influenza.⁴

Gout, according to Semon and Watson Williams, is, comparatively speaking, the most fertile source of pharyngeal hemorrhage.

Bleeding from the surface of the laryngeal mucous membrane must be distinguished from submucous hemorrhage.

Blood clots may not be expelled but remain lodged in or near the vocal cords, so as to simulate the appearances of an angioma, carcinoma or a soft fibroma.^{5 6 7}

Symptoms.—A slight clearing of the throat is often all that precedes the patient's discovery of blood in his mouth. If it comes in any quantity the expectoration is accompanied by the peculiarly sickening and depressing taste and smell of blood.

When no trace of a leaking vessel is visible, the case should be treated as one of early pulmonary tuberculosis, particularly if any suspicious indications are present—fatigue, anorexia, loss of weight, evening rise of temperature, anemia, neurasthenia, or a history of pleurisy.

In my experience as a laryngologist, hemoptysis is due to pulmonary tuberculosis in the great majority of cases. When it has been absolutely necessary to demonstrate this, I have done so by passing an endoscope and viewing the blood coming up from one or other bronchus.

It is, indeed, quite rare for blood in any quantity to "come from the throat" except in well marked local lesions. It is easily, and not infrequently, produced by malingerers making suction on their gums.

Treatment.—Treatment depends on the discovery of the source of bleeding. The local bleeding may require adrenalin, hamamelis (Pond's Extract), catechu, or other astringents. The galvanocautery, if at hand, is often the speediest remedy. When large vessels are eaten into by cancer or abscess, it may be necessary to tie the external or common carotid.

The administration of lactate of calcium will increase the coagulability of the blood. A hypodermic injection of morphia, gr. 1/6 to 1/4, with atropin, gr. 1/200 to 1/150, is one of the readiest, quickest and most reliable remedies.

In all cases the patient should be ensured complete rest and fresh, cool air. Alcohol and hot fluids should be forbidden. Solid food is not necessarily avoided. Excitement and fear must be guarded against. The sucking of ice, a weak spray of adrenalin, and small doses of opium may be indicated.

When it is even suspected that the blood is of pulmonary origin, we should strongly advise a sojourn in a tuberculosis sanatorium. There the patient will learn how to raise and maintain his resistance, and the case records of such institutions

would then have less frequent occasion to write: "The patient had a blood spitting some years ago but took no notice of it, as a laryngologist told him it came from the throat."

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XIV.

THE SYMPTOMATOLOGY, DIAGNOSIS AND TREATMENT OF LARYNGEAL TUBERCULOSIS.

BY FRANK R. SPENCER, A. B., M. D., F. A. C. S.,

BOULDER, COLO.

The history of laryngeal tuberculosis is an interesting portrayal of scientific achievement in human progress for the past 150 years. It is in keeping with the progress of scientific medicine and surgery, the foundation of which was laid before our own time; but the greatest progress has undoubtedly been made during the lifetime of those older laryngologists now living. The advancement of laryngology came with the invention of the laryngoscope, by Manuel Garcia, in 1855.

Another great impetus was given to the clinical examination of the larynx, since 1900, by Killian when he devised the suspension laryngoscope, and by Lynch when he improved and perfected Killian's instrument. Lynch made the suspension laryngoscope a useful and practical instrument. He also added a complete set of direct laryngeal instruments for examinations and operations. The direct laryngoscopes of Jackson and Mosher, with the more recent one devised by Haslinger, have been valuable aids to diagnosis and treatment.

It may then be justly said that the achievements in laryngology are keeping pace with advances in other special fields, such as internal medicine, general surgery and roentgenology. It is this progress which should enable us to make an early diagnosis of laryngeal tuberculosis and institute proper treatment, if we hope to lessen the high mortality of late laryngeal tuberculosis.

Prevention of all forms of tuberculosis should receive careful consideration from the medical profession, if we expect to materially reduce the high death rate from this disease. Public health journals are doing a great deal of good in this direction. Hygeia is reaching many homes, but it should be in every home.

The prevention of pulmonary tuberculosis can best be carried out by systematic and pleasant exercise in the fresh air, and especially in the sunshine. Fresh air should be provided in every public and private building and particularly in the sleeping room. Good wholesome food for children and adults builds up resistance to disease. Proper hours for rest and recreation are very essential. Cathell¹ says, "rec-reation is re-creation." Avoid exposure to patients with a cough. Patients should be requested to cover the mouth with a handkerchief whenever they cough or sneeze. A sputum cup must be used for all sputum.

Prevention of the laryngeal form of the disease, so far as our present knowledge will permit, can only be accomplished by an early diagnosis of the pulmonary tuberculosis, and with proper treatment. It has been variously estimated that from 45 to 90 per cent of all patients with advanced pulmonary tuberculosis develop laryngeal tuberculosis as a complication, for from one month to many months before death.

Subjective symptoms are often mild and attract very little attention, either from the patient or a physician, in the early stage of the laryngeal disease. The patient may be hoarse in the morning after getting up, or during the day, if he has been coughing, talking, smoking, etc. Rest usually relieves the hoarseness or mild home remedies do. The attacks of hoarseness may occur more and more frequently. Aphonia may be an occasional additional warning, and if unheeded, may become complete and permanent. Tickling, a thick or full feeling, constriction, general irritation of the throat, followed later by dyspnea, dysphagia; or, even odynphagia may all annoy the patient. However, dysphagia, and especially odynphagia, are late symptoms, and dangerous symptoms. To wait for these danger signals is to wait, oftentimes, until it is too late to save the patient's life.

The objective symptoms may consist of a slight anemia, with mottling of the laryngeal mucosa, chiefly on account of the secondary anemia from which most patients with pulmonary tuberculosis suffer. If the patient is coughing a great deal there may be hyperemia or congestion caused by the irritation of the larynx. These are in reality pretuberculous symptoms and should be so regarded, but they should not go unheeded.

Infiltration of the posterior laryngeal wall, and particularly the interarytenoid sulcus, is a frequent and an early symptom. Later the infiltration may extend to one arytenoid. Tuberculosis manifests itself early in the posterior half of the larynx far more frequently than in the anterior half. Occasionally infiltration involves the cushion of the epiglottis or the aryepiglottic folds.

Ulcers, whether acute or chronic, superficial or deep, are later and more serious symptoms. These may produce very little discomfort at first, but are likely to become a serious menace to the patient's welfare on account of the dysphagia and odynphagia which they soon cause.

Tuberculomata or tuberculous granulomata may be easily mistaken for papillomata, because of the similarity clinically. Usually there are other tuberculous lesions in the larynx which should attract attention.

Edema is nearly always a late symptom. The term pseudo-edema is better, because the condition is not a true edema, as suggested by Sir St. Clair Thomson.² "Ashen gray" appearance is also used to describe this lesion. Whenever tubercles are large enough or numerous enough to interfere with the circulation of the larynx, edema results. It generally indicates an advanced laryngeal tuberculosis.

Fibrosis represents nature's effort to cure, or at least arrest, the disease. This effort should be encouraged, because we know that it often results in permanent benefit to the patient. Spontaneous healing of incipient pulmonary tuberculosis, and even of laryngeal, is not unknown by any means.

Perichondritis, chondritis and necrosis are all late symptoms. If confined to the free edge of the epiglottis they offer a better opportunity for treatment. When the arytenoids are involved the outlook is very serious, and likely to be rapidly fatal.

The diagnosis must be based, from the laryngeal viewpoint, on the previously described lesions. However, the examination is not complete until an internist, skilled in chest work, has made a general physical examination. This should be supplemented by roentgenograms of the chest, urinalysis, white and differential blood count, at least one blood Wassermann test, sputum examinations, microscopic examination of laryngeal tissue in very doubtful cases, such as tuberculoma, etc. How-

ever, biopsy is very rarely a justifiable, or even a necessary procedure, for the diagnosis of laryngeal tuberculosis. The diagnosis can usually be made rather easily without biopsy.

Differential diagnosis must of necessity take into consideration at least the following diseases: Syphilis, carcinoma, actinomycosis, lupus, chronic laryngitis, and, occasionally, such a rare disease as leprosy.

Syphilis is so common at the ages of 20 to 45, just as laryngeal tuberculosis is, that it should always be borne in mind. The disease is so variable in its lesions, and can easily duplicate the macroscopic appearances of tuberculosis.

Carcinoma is more frequent past 50 years of age, and in patients who haven't tuberculosis, although the two diseases may occur as dual lesions in the same larynx. Dr. L. W. Dean of Iowa City and Dr. Joseph C. Beck of Chicago have each told me they have seen tuberculosis, syphilis and carcinoma present in the same larynx and at the same time. The three lesions at different periods of the life of any patient would not be so unusual. Carcinoma is more frequent in the anterior half of the larynx, especially early, than tuberculosis. Biopsy is more important in doubtful cases of carcinoma than in tuberculosis or syphilis. However, Mackenty³ has been able to repeatedly diagnose early cancer without the aid of biopsy. His long experience with this disease and his mature judgment make him much more expert than the average laryngologist can hope to be with carcinoma.

Actinomycosis is most frequent in farmers and ranchers. The laryngeal lesion may be accompanied by lesions in the mouth, pharynx or nose. The lungs are usually normal, and tuberculosis of other organs of the body is not likely. Biopsy reveals the ray fungus.

Lupus is rare in this country but common in Europe. The disease is very slow in development. The eruptions about the face are a common accompaniment of the laryngeal disease and often precede the laryngeal form. The lungs are rarely involved.

Chronic laryngitis yields usually to treatment and is often present without disease in other parts of the body. Excessive use of the voice is a frequent cause, hence the importance of rest for diagnosis.

Leprosy is extremely rare. The lesions are usually not limited to any one organ. The finding of the lepra bacillus of Hansen is regarded as diagnostic. The tubercular form of the disease, should it occur first in the larynx, might be very difficult of diagnosis. I have seen only a very few cases, but the multiple lesions in the skin help in making the diagnosis.

The accompanying table may help in making the differential diagnosis :

TABLE OF DIFFERENTIAL DIAGNOSIS.

Signs, Symptoms and Tests	Laryngeal Tuberculosis	Syphilis	Carcinoma	Actinomycosis	Lupus	Chronic Catarrhal Laryngitis
Hoarseness	Early	Early	Early	Frequent	Frequent	Early
Aphonia	Late	Late	Late	Rare	Rare	Rare
Early Pain	Rare	Frequent	May occur	Rare	Rare	Very rare
Late Pain	Frequent	Frequent	Very often	Rare	Rare	Rare
Dysphagia	Early	Early	Early	Rare	Rare	Late
Odynphagia	Late	Late	Early	Rare	Rare	Late
Bacteria	Tubercle Bacilli	Spirochaeta Pallida	?	Ray fungus	Tubercle Bacilli	Mixed Infection
Presence of Sputum	Frequent	Rare	Rare	Rare	Rare	Frequent
Lungs	Pulmonary T. B.	Negative	Negative	Negative	Negative	?
Blood Wassermann	Negative	Positive	Negative	Negative	Negative	Negative
Spinal Fluid Wassermann	Negative	Positive	Negative	Negative	Negative	Negative
Roentgenogram of Lungs	Positive	Negative	Negative	Negative	Negative	Negative
Biopsy	Tubercles	Gummata	Carcinoma	Ray fungus	Granuloma	Chronic Inflammation

Treatment consists of several well established methods as follows:

Rest of the larynx can be accomplished best by absolute silence. However, this may not always be required. Whispering, if restricted to short periods of the day, may be sufficient. It is often much less depressing, and can be better regulated in a sanitarium or hospital than in the patient's home. To restrict a patient to a scratch pad and pencil at first may meet with bitter resentment.

The solar laryngoscope often offers one of the very best methods of arrest, or even cure, of the tuberculous larynx. With a little practice it is comparatively easy to use. The alloy of magnesium and aluminum reflects the ultraviolet, actinic or chemical rays of the sun into the larynx, whereas, glass absorbs these rays. Forster and Chapman⁴ have helped to perfect such a laryngoscope. Exposure of the larynx for ten seconds daily

the first week and gradually increasing the length of each treatment by five seconds each week until the treatment consumes ten minutes, if tolerated well, seems best. Some patients stand exposures of 20 minutes daily, but this is the exception. Over exposure, to begin with, sunburns the larynx.

For many years the cautery has occupied a very important place in the treatment of laryngeal tuberculosis. Infiltrations, ulcers, whether superficial or deep, areas of pseudoedema and the base of a tuberculoma, following its surgical removal, all may require cauterization. Dean⁵ and Arrowsmith⁶ often favor the direct method by suspension. Sir St. Clair Thomson² and Greene⁷ prefer the indirect method. The latter is often easier for the patient, but the method of procedure selected should be the one best suited to a particular patient.

Patients usually tolerate cauterization better several hours after a meal. Even a very light meal just prior to cauterization is often vomited; an empty stomach is best for most patients. Gargles and sprays are not indicated.

Perfect local anesthesia is very necessary for successful cauterization. If the patient gags easily, is nervous and apprehensive, the laryngologist's work is made very difficult. A 20 grain dose of sodium bromid administered one hour beforehand has a quieting effect. Morphin sulphate, grains $1/6$ to $1/4$, with atropin sulphate, grains $1/200$ to $1/150$, given hypodermically one-half hour before, are often better. Personally, I prefer not to use atropin because of the greater danger of cocain poisoning, if atropin and cocain are used together. The final preparation can best be carried out by spraying the pharynx, fauces and larynx with a 5 per cent aqueous solution of cocain. After five minutes the larynx should be further anesthetized by applying powdered cocain on a cotton wound applicator, or, better, by dropping a 10 per cent aqueous solution about the rim of the larynx from a laryngeal syringe. Brunings'⁸ syringe is admirably adapted to this work. After several minutes' wait a few more drops of the same solution may (with the aid of the laryngeal mirror), be dropped into the center of the larynx while the patient says "ah." This process may be repeated after a few minutes, and the solution dropped on the arytenoids, interarytenoid region, and cords,

particularly on the lesion to be cauterized. A few drops of 1/1,000 adrenalin chlorid solution should be added to each of the cocain solutions to limit the local action of the cocain and lessen absorption into the general circulation. The final preparation should not require more than twenty minutes.

After the cocainization is complete, the direct laryngoscope of Jackson or Mosher may be introduced, or the patient may be suspended with Lynch's suspension laryngoscope. Haslinger's directoscope is also very useful. Any one of the preceding will give a direct view. Thomson² and Greene³ prefer the indirect method. I have done much of my work by the indirect method, but I use both the direct and indirect.

Rather long, sharp cautery points are best. These should be heated to a white heat and plunged deep into the lesion. If the point is red, the surface is seared without reaching the tubercle. Fibrosis is the purpose of cauterization, in order to destroy the tubercle ultimately, not total destruction at the time of the cauterization. The cautery electrode should be introduced into the lumen of the larynx and held near the tubercle. The current should be turned on, and the platinum point pushed into the tubercle as soon as the point is at a white heat. At a white heat it is easily used and withdrawn without adhering to the burned tissue. Two or three areas can usually be cauterized at one time, or even more, if the patient behaves well. If the patient's general and pulmonary condition will not permit much to be done, or if the patient is not cooperative it is far better to do too little rather than too much. Normal areas of the larynx should be carefully avoided.

The joints about the arytenoids should be shunned. Subsequent fibrosis with contraction will produce ankylosis and result in fixation of one or both cords.

Hemorrhage, either at the time or subsequently, may occur if the platinum point is used only red hot, because the eschar tends to adhere to the red point and leave a bleeding surface. The platinum point should be used at a white heat, because of the ease with which the point can be plunged into a tubercle and withdrawn, without detaching the eschar and without causing hemorrhage. Occasionally a secondary hemorrhage may occur when the slough separates on the tenth to the fourteenth day. This, however, is rarely severe.

Infection following the use of the cautery is extremely rare. This is doubtless due to the destruction of infectious organisms by the cautery and the sealing of the lymph and blood vessels by the eschar.

Very little, if any, after care is required. Mild palliative remedies are useful for patients who demand that something more be done. Dropping a few cc. of 1 per cent menthol in glymol into the larynx, from a laryngeal syringe, allays irritation. Narcotics are rarely required.

The reaction following the use of the electric cautery is, as a rule, not severe and patients usually recover rapidly during the subsequent ten to fourteen days. There is very little or no pain, either at the time or subsequently. Patients say the larynx feels thick or that it feels full. A feeling of suffocation could doubtless be produced by extensive cauterization at one time, but this must be avoided. Subglottic edema, especially in adults, is not likely following the judicious and careful use of this instrument. Rest in bed is best for a few days.

The cauterization can easily be repeated after a few weeks or months, depending upon the indications.

The injection of tuberculin directly into the larynx is of benefit in a very few selected cases. I¹⁰ have used this method of treatment, but cannot recommend it in preference to the cautery and other well established methods of treatment. Sluder's syringe, with a long, curved needle for the indirect, and a long straight needle for the direct injection of tuberculin is useful. The needles were made, under my direction, for use with Sluder's syringe. Several general, local and focal reactions must be avoided. Mild local and focal reactions in the larynx are desirable. Very much edema at the point of injection is quite undesirable. The administration should begin with 1/10 of a milligram of O. T. The dose should be gradually increased if the preceding one has been well tolerated. An interval of two to six weeks between injections is an additional safeguard. The injection should be made at the point of greatest swelling after cocaineizing the larynx. The point of the needle should penetrate deep enough to reach the center of the tubercle, if possible. A submucous injection is less desirable.

Small doses and in small volumes are best. The volume should not exceed $\frac{1}{2}$ to 1 cc. in order to avoid edema at the point of injection. A large dose is apt to produce a severe reaction in the larynx with more or less severe general reaction. Prime reasons for making the injection directly into the larynx are: First, to place the tuberculin where it is needed most, and second, to avoid the general reaction.

The objection may easily be offered that the effect on the larynx will not be materially different from what it would be if the tuberculin were injected subcutaneously. However, this is not a just criticism. If the laryngeal injection can be made directly into the center of a tubercle, the maximum therapeutic effect will occur at the point of injection. The absorption from the caseated center should be slow, with the maximum local therapeutic effect and with a minimum general effect. Since it is the local action we want in the larynx and not the general, this method of treatment in selected cases is of value.

If it were desirable, in any given case, to build up the patient's general resistance to tuberculosis first, the subcutaneous injection of tuberculin would be the method of choice, provided tuberculin is to be used at all. However, we occasionally see patients who are making good progress, so far as their general health and pulmonary tuberculosis are concerned, and who have only a slight involvement of the larynx, but in whom the laryngeal disease is advancing slowly. In such cases injection of tuberculin into the tuberculous area of the larynx certainly seems to have a beneficial effect.

By making the injection in the larynx, instead of subcutaneously, the site for the local reaction can be selected in the larynx with the advantage of combining the local with a mild focal reaction. It is the local reaction which seems to produce the improvement by local immunization and local stimulation. It is, of course, impossible to tell how much of the reaction is focal and how much is local. I have tried (by using small doses), to avoid anything more than a mild focal reaction. The dose may be gradually increased up to 100 milligrams, but it need not exceed this amount.

In conclusion, several points deserve consideration:

1. The importance of an early diagnosis of pulmonary tuberculosis and proper treatment can scarcely be emphasized too

much. It is the best way to prevent laryngeal tuberculosis and offers the best opportunity for an early cure.

2. The early diagnosis of laryngeal tuberculosis is next in importance.

3. Having made the diagnosis, some form of rest for the larynx must be resorted to.

4. The solar laryngoscope offers an excellent method of treatment.

5. The cautery is often required for lesions which have resisted all other forms of treatment.

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PHYSICIANS' BUILDING.

XV.

HEMORRHAGE INTO THE VOCAL CORD: CURED
BY ELECTROLYSIS.

BY GREENFIELD SLUDER, M. D.,

ST. LOUIS.

The following case is reported because of its rarity.

P. D., age 25, became my patient in 1901. He was a lyric tenor in the grand opera of those days. He stated that on an occasion six months before, when he had a cold in his throat and was hoarse, while making an effort to sing "over" his cold, he was suddenly seized with something that stopped his voice. He had been treated during this six months by several specialists, but he still remained hoarse and could not go back on the stage. Examination showed the left cord of greater



volume than the right. I took the seizure to have been a hemorrhage into that cord. For several months I kept him on treatment consisting of dilute iodine and silver nitrate, 2 per cent, instilled into the larynx. His voice remained unchanged. I then tried an incision through the vocal cord, using the technic of Krause,* making an effort to divide the blood vessels and cause a subsequent shrinkage. This accomplished something. The volume of the cord was a little smaller. The incision was made through the entire thickness of the cord, from top to

*Die Erkrankungen der Singstimme; ihre Ursachen und Behandlung. Nach einem Referat vorgetragen auf dem zwölften internationalen medizinischen Kongress zu Moskau. Von Prof. Dr. H. Krause, Berlin, 1898. Verlag von August Hirschwald.

bottom and from the vocal process to its origin in front. This procedure was repeated four times, the last time without any subsequent shrinkage. Knowing from the study of dermatology that electrolysis would produce shrinkage, I attached the naked knife, which was of lancet shape, to cut forward and backward, to the electric battery, consisting of a number of cells, such as were used by the dermatologists of those days, the cells being made with chlorid of silver. With one hand in a glass of water, to which the positive electrode had been attached, I increased the number of cells until I got pronounced electrolytic effect shown by effervescence around the knife blade which was in the cord. The blade was left in the cord and the electric action continued for ten minutes. Under cocain and by the indirect method the procedure was painless and was followed by a definite shrinkage in the volume of the cord. At the end of a month the procedure was repeated again and the electric action given for five minutes. This was followed by a shrinkage of the line of the cord, an appearance that was indistinguishable from the normal. I waited another month and told him to try his voice, and to our delight it seemed practically normal. I instructed him to proceed slowly but to begin using it. After six weeks he was delighted to tell me that his voice was all right again. I then told him to take a place in some small part in the opera and see whether he could work back into power of endurance. This he did in another city. He remained on the stage for three months, when he was stricken with pneumonia and died. I, of course, did not get an autopsy, but his friends said that his voice had been all right.

XVI.

POSTERIOR CICATRICAL STENOSIS OF THE LARYNX.

BY GREENFIELD SLUDER, M. D.,

ST. LOUIS.

This case has been unique in my experience, nor have I seen another case reported of similar nature. The fact of its being so long in the past seems to me to add to its interest.

Mrs. G. B. M., age 50, consulted me in the autumn of 1899. At that time she gave a history of a healing pulmonary tuberculosis, and for five years previous she had been treated for "trouble" in her throat. Consultation with her physician, Dr. George B. Smith, revealed the fact that five years previously



she had had an ulcer in the larynx which extended around from one vocal process to the other. She lived in the country and could not be sent to specialists in the city, so he applied lactic acid. The ulcer had slowly healed.

When she came to me the larynx appeared completely healed and the scarring process had contracted until the posterior third of the glottis was obliterated and the vocal processes approximated. Temperature was normal, with every indication that the pulmonary lesion had healed. Realizing that something had to be done at once to gain breathing space, I incised the scar in three directions, one from the vocal process straight back and two from the vocal process at an angle of 45 degrees outward and backward. This was done by the indirect method with a naked knife under cocain. The result was

an opening of the glottis to what appeared to be normal. It was an equilateral triangle. The three incisions were made because the first one, in the midline alone, had no effect. The bleeding was so little as to be practically nothing. She remained in my charge about two weeks and returned home. She came back to me in two months. Inspection at that time showed the wounds to have healed and a scar contracted to about approximately one-fifth the normal glottis. Her general condition was vastly better. I then incised the scar through the same lines, in the same way, reestablishing what appeared to be a normal glottis, and again waited for this to heal. In all, the larynx was incised as above described five times at intervals of from two to three months, each time gaining a little more breathing space, until finally the glottis appeared to be normal. She continued to report from time to time. Finally, the incisions were repeated once more a year and half later, because of some narrowing which had slowly taken place. The end result in this case gave a glottis apparently seven-eighths of its primary caliber. The last observation was made in 1918 and the glottis had not narrowed.

XVII.

CONTACT ULCER OF THE LARYNX.

BY CHEVALIER JACKSON, M. D., Sc. D., LL. D.,

PHILADELPHIA.

Superficial erosions are not uncommon in chronic laryngitis, but there is a shallow ulcerative lesion that is deserving of a special designation and a particular recognition, because it is so frequently either overlooked altogether, in the milder cases, or mistaken for tuberculous or malignant disease in the case of larger lesions. A series of pathologic and clinical studies will be presented subsequently; only a brief résumé of prominent features will be presented here.

Definition.—Contact ulcer is the name I have given to superficial ulceration occurring on one or both sides of the larynx posteriorly, the ulcerated surface coming in contact on phonation with that of its fellow of the opposite side, the latter being ulcerated or not, according to whether the ulcer is monolateral or bilateral.

Incidence.—The lesion is not a common one, inasmuch as I have seen only 127 cases in forty years.

Etiology.—The location and the evident contactual pressure seen in the mirror during phonation seem to indicate vocal abuse as the chief etiologic factor. This is corroborated by the fact that the condition is observed in persons using the voice excessively, and it is further corroborated by the observation that cure is usually impossible without vocal rest. Most of the cases have been seen in conjunction with chronic laryngitis, which would justify the inference that chronic inflammation of the laryngeal mucosa is also a cause, or at least that the ulcer and the chronic laryngitis are both dependent upon a cause common to both. Many of the patients complained of waking with choking, strangling cough at night, relieved after secretions were coughed out of the trachea. This suggested leakage into the larynx of oral secretions that should drain

away downward into the esophagus. Such secretions would carry oral infection. This symptom was not present in all cases, however.

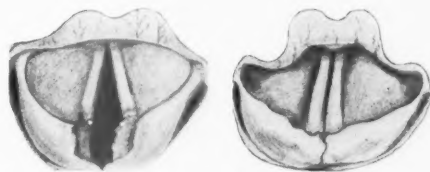
Pathology.—Histologic examinations by Ralph Duffy, Joseph H. Barach, Ernest Willetts, Ernest Funk, W. M. L. Coplin, B. F. Crawford, Herbert Fox and Carl J. Bucher in the various cases in which specimens have been taken to exclude malignant and tuberculous disease have all shown the usual histologic characteristics of chronic inflammation with ulceration. No specific histologic characteristics have been found so far. Bacteriologically the lesion has been found to be most frequently associated with Vincent's organisms, but in many typical cases these organisms were absent. While the ulcer is not deep, the vocal process of the arytenoid cartilage, because of its superficial position, is often involved and it becomes necrotic.

Symptomatology.—Hoarseness is present in most cases, though it may be slight. Pain or at least discomfort in the larynx is usually present, though it may be overlooked by stolid individuals. In a few cases it was severe and radiated to the ear of the ulcerated side. The symptoms are usually marked by those of the accompanying chronic laryngitis. Choking and strangling at night from overflow of oral secretions into the larynx was present in a number of cases.

Diagnosis.—Exclusion of cancer and tuberculosis is called for in all cases. In many cases the extremely superficial character of the ulcer, rather an erosion or loss of epithelium than a true ulceration, taken together with the free mobility of the arytenoid, renders it advisable to await the result of a regime of absolute silence and omission of all irritant forms of treatment before taking a specimen. If, however, the ulcer persists after a month or two, biopsy is advisable. The utmost care and precision are required to avoid injury to the cricoarytenoid joint. When necessary for diagnosis, the excision usually has the added advantage of promoting cure of the ulceration. At biopsy the necrotic vocal process of the arytenoid cartilage is often found in the bed of the ulcer. No harm is done if a very small amount of the extreme tip of the process is removed, and, in fact, healing may follow.

Laryngoscopic Appearances.—In the mirror the ulcer is seen on edge, involving the almost perpendicular internal surface of the arytenoid cartilage. On inspiration the ulcer is exposed to view, but on phonation its internal surface is in contact with the internal surface of the opposite arytenoid eminence, which may or may not be ulcerated. Usually the thickened border of nonulcerated mucosa at the upper margin of the lesion is seen, but the ulcerated surface is not. In some instances, the border forms a tiny roll rim around the ulcer, and the vocal process of the opposite arytenoid fits into the tiny bowl-like lesion. In some instances in the bottom of the bowl is found the denuded vocal process of the ulcerated arytenoid. The usual site of the ulcer is the area in the neighborhood of the vocal process.

Prognosis.—Under mild, gentle, careful treatment, in almost all cases ultimate recovery has followed; but the progress has



been rather slow in most instances. Energetic ill advised treatment is usually followed by edema, loss of cartilage, impairment of the arytenoid joint, and increased hoarseness. In one patient sent to us, extensive cauterization had caused perichondritis, with acute edematous stenosis, followed by chronic cicatricial stenosis. If the patient persists in vocal abuse, prognosis as to cure is unfavorable. In two such cases, cancer ultimately developed. In two other cases, in patients with pulmonary tuberculosis, the ulcers ultimately became tuberculous.

Treatment.—The fundamental requirement is absolute silence, the patient writing every word he has to say. Even whispering should be forbidden. All irritant applications, such as silver nitrate, or any other medicaments on a swab, are

contraindicated. If after two months of silence have failed to cure the ulcer, it is better to excise it with great care and precision. Care should be taken to avoid damage to the crico-arytenoid joint and to avoid removal of a normal vocal process. If the vocal process is necrotic, removal of a small portion may promote healing. When there is a thickened rim of epithelialized mucosa around the ulcer, careful nipping off of this rim with small cupped forceps usually promotes healing in from the edges. After operation, residence at the seashore promotes healing. The ozone, chlorin and vapor of salt water, inspirated twenty-four hours out of the twenty-four, is of great benefit, not only to the ulceration, but also to the accompanying chronic laryngitis.

XVIII.

ASPIRATION OF BLOOD INTO THE LARYNX AND TRACHEA DURING TONSILLECTOMY UNDER LOCAL ANESTHESIA: A CONTRIBUTION TO THE ETIOLOGY OF LUNG ABSCESS.

By SAMUEL IGLAUER, M. D.,

CINCINNATI.

The frequency and seriousness of posttonsillectomy lung abscess make it imperative to study all the factors which may be concerned in its production. Recent observations and experiments have thrown much light on the etiology of this dread complication.

According to the estimate of Moore,¹ pulmonary abscess occurs about once in from 2,500 to 3,000 tonsillectomies. The figures were obtained in response to a questionnaire in which 202 cases of pulmonary abscess were reported following operative work, chiefly tonsillectomy, about the upper respiratory passages. Thirty-nine, or about one-fifth of all the cases followed operations under local anesthesia. From the literature Cutler and Schlueter^{2,3} assembled 1,908 cases of abscess of the lung. Of these 515, or 29.6 per cent, were postoperative, and 14.6 per cent, approximately one-half, followed tonsillectomy. According to Hedblom,⁴ of 146 postoperative lung abscesses observed at the Mayo Clinic, 48 (33 per cent) followed tonsillectomy. Fourteen (9.5 per cent) extraction of teeth, and 38 (26 per cent) laparotomy. With a few exceptions these operations were performed under general anesthesia. Lord⁵ reviewed 227 cases of lung abscess. Of these 96 were postoperative, and of the latter group 78 (81.4 per cent) followed operations about the upper respiratory tract.

It has long been recognized that operations about the mouth and pharynx carry with them the potential danger of pulmonary complications, and since tonsillectomy is the operation most frequently performed in this region of the body, this

particular operation has borne the chief burden of being accountable for these serious sequelæ.

The older generation of surgeons was of the opinion that these pulmonary complications were due to the irritation of the anesthetic (ether pneumonia) or to the aspiration of blood saliva and infected material from the pharynx.

In recent years, however, many have attributed the pulmonary complications to embolism rather than to direct aspiration from the operative field.

EMBOLIC THEORY.

Clinical: Cutler and Hunt⁶ have made careful clinical and X-ray studies of patients suffering from postoperative pulmonary complications. They found that such complications, chiefly in the form of bronchopneumonia, are not infrequent after operations, especially laparotomy; in some instances, laparotomy under local anesthesia. They conclude that the majority of these pulmonary lesions are of embolic origin and "that there is an increasing mass of evidence demonstrating the frequency of postoperative embolism and its relation to complications."

Experimental: On the experimental side, Schlueter⁷ and his associates were unable to produce lung abscess in dogs by the intrabronchial injection of bacteria or tonsil tissue. On the other hand, they induced lung abscesses in their animals when they inserted an infected artificial embolus into the opened jugular vein. This embolus consisted of a sausagelike vein segment, filled with virulent cultures of bacteria of various kinds. In contrast to this, they found that freeing simple infected and uncovered clots into the venous circuit usually resulted in a general pneumonitis rather than in a walled off abscess.

Fetterolf and Fox⁸ studied the reaction of the paratonsillar tissues in dogs after tonsillectomy. Around the tonsillectomy wound they found hemorrhages into the tissues, areas of necrosis, bacteria and injured vessels closed by coagula. They consider it quite possible that small emboli from the tonsillectomy wound may give rise to septic infarcts in the lung.

ASPIRATION THEORY.

Experimental: In 1898 Hoelscher⁹ made a careful study of the lungs of etherized animals and found that the buccal secretions, previously stained, were constantly present in the tracheobronchial tree and even in the alveoli of the animals. The ether alone produced no ill effects, but the aspirated secretions set up a pneumonitis.

The recent experiments of Lemon¹⁰ on etherized dogs showed conclusively that dyes, or barium suspension, slowly introduced into the mouths of the animals were drawn into the lower respiratory tract in any posture, short of the full Trendelenburg position. Lemon concludes that "postoperative pulmonary infections may be explained, in part at least, as a result of infection carried to the lungs in oral secretion which has been aspirated by the force of the air current when the chest expands, in patients whose protective mechanism has been disturbed."

It remained for Smith¹¹ to successfully induce lung abscess in mice, guinea pigs and rabbits by the intratracheal route. After etherizing the animals and exposing the trachea, he inserted a needle into the trachea and slowly injected bloody material obtained from about the teeth of patients suffering with moderately severe pyorrhea. Approximately 20 per cent of the animals developed lung abscess, 50 per cent died of pneumonia and 30 per cent remained well. Smith pointed out that lung abscesses in man usually contain two or more of the following organisms: spirochetes, fusiform bacilli, cocci and vibrios, "an assortment of organisms found in the tonsillar crypts and around the teeth," and concluded that infected material from the mouth accounted for most cases of pulmonary abscess.

In the January, 1928, *Archives of Surgery*, Crowe and Scarff¹² report on the production of typical lung abscesses in dogs by the introduction through the bronchoscope of small pieces of cotton saturated with fresh scrapings obtained from pyorrhea cavities of patients.

In the same journal, Allen¹³ reports on the successful production of lung abscesses in the dog by injecting warm pus from a lung abscess or minced tonsil tissue into a bronchus.

after which the same bronchus was immediately ligated with silk (thoracotomy).

From this group of experiments it would appear that obstruction of the infected bronchus with optimal temperature for the bacterial invaders constitute important factors in the development of lung abscess by the endobronchial route.

Clinical: Myerson^{14 15} bronchoscope 200 children immediately after tonsillectomy, performed under ether anesthesia, and found blood in the tracheobronchial tree in 155, or 77.5 per cent of all the cases. This aspiration occurred despite the fact that he employed most of the usual devices, such as posture and suction, to prevent the entrance of the mouth contents into the trachea.

Daily and Daily¹⁶ found blood in the tracheobronchial tree in 78 of 100 patients bronchoscope immediately after tonsillectomy under general anesthesia. They also performed esophagoscopy in most of these patients, and frequently found blood in the stomach. They consider light anesthesia, with an active swallowing reflex, important in preventing aspiration. They also examined three cases operated upon under local anesthesia. In two of these patients the larynx was cocaineized at the beginning of the operation. Blood was found in the lungs and esophagus in one of the cocaineized patients. The two remaining patients had blood in the esophagus, but not in the lungs.

At the Cincinnati General Hospital my associates, Doctors Vail, Goodyear, King and Smith have bronchoscope a total of 100 children immediately after tonsillectomy and adenoidectomy, and have found blood in the trachea or bronchi, or both, in 40 per cent of all our cases, a figure considerably lower than those of Myerson and the Dailys. (Our cases will be reported in greater detail at some future time.)

While by far the greater number of posttonsillectomy abscesses have occurred after general anesthesia, a considerable percentage has ensued after local anesthesia. It has been assumed by some of those opposed to the aspiration theory that blood and mucus do not enter the trachea during tonsillectomy under local anesthesia and that, therefore, the aspiration theory does not apply in these cases.

OBSERVATION OF LARYNX AND TRACHEA AFTER TONSILLECTOMY UNDER LOCAL ANESTHESIA.

In order to investigate this matter, I decided to examine a series of patients immediately after tonsillectomy performed under local anesthesia, to determine whether or not blood had entered the larynx and trachea during the operation. Fifty adult patients were examined (one girl of 16).

The tonsils were anesthetized by injecting procain (1 per cent) with epinephrin into the pillars and paratonsillar tissues in the usual manner. Cocain was not applied, in order to exclude any possible anesthesia of the pharynx and larynx. Twenty-one of the patients received a preliminary hypodermatic dose of morphin, grain 1/6, with atropin, grain 1/150. The patients were all operated upon in the upright posture and were encouraged to expectorate any blood that collected in the mouth. The dissection and snare technic was employed. Sponges on sponge holders and clamps were used when necessary.

As soon as the bleeding ceased, the laryngeal mirror was introduced and the larynx and upper trachea were inspected. It was found that owing to the infiltration anesthesia of the fauces, laryngoscopy and upper tracheoscopy could be carried out with unusual ease in practically every patient.

Observations were made as to the presence or absence of blood and mucus, and to what extent and in what quantity it had descended into the trachea.

Blood was seen in both the larynx and trachea in fourteen cases (28 per cent) and in the larynx alone in two cases; and in the trachea alone in five cases, giving a total of nineteen cases (38 per cent) with blood in the trachea.

The upper trachea contained blood varying from mere staining to amounts estimated at twenty drops. The maximum distance at which blood could be seen was about one-third the length of the trachea.

These observations prove that, contrary to the general belief, blood very frequently enters the larynx and trachea in tonsillectomy under local anesthesia. The blood in the larynx probably has no bearing on the development of lung abscess, but the blood, mucus and crypt contents in the trachea may

give rise to pulmonary infection under suitable conditions. Such predisposing factors would be the activity of the cough reflex, the position assumed after operation, existing or previous bronchopulmonary infection, and the virulence and quantity of the organisms in the inspired material. Under normal conditions the trachea and bronchi are cleared of foreign substances by coughing, by the action of the ciliated epithelium and possibly by peristaltic waves, as claimed by Bullova and Gottlieb.¹⁷ As observed in several of these patients, the inspired blood is very tenacious, but a small part can be expelled by coughing. As a rule, patients refrain from making any forced movements of the throat after tonsillectomy, and by suppressing the cough reflex favor the retention of aspirated material.

Ochsner and Nesbit¹⁸ have approached this problem in another manner. In a series of five cases following peritonsillar infiltration for tonsillectomy, the patient was placed behind the fluoroscopic screen and given iodized oil. Upon attempting to swallow, the oil went the "wrong way," and in every instance was seen to enter the trachea and bronchi. Ochsner concludes that "this observation removes the one objection to the aspiration theory of abscess of the lung following tonsillectomy."

It occurred to me that one objection to this experiment lay in instructing the patient to swallow, whereas the patient is usually urged to expectorate during tonsillectomy. I therefore modified the experiment and slowly injected lipiodol (total 5 cc.) into the tonsillar fossæ immediately after tonsillectomy without giving the patient any instructions. Of the five patients treated in this manner, one had lipiodol in the right bronchus and the stomach, a second had about two drops in a bronchus, and the remainder of the oil in the stomach, while the other three patients had all the lipiodol in the stomach. These observations show that under ordinary tonsillectomy conditions blood enters the tracheo-bronchial tree in some but not all cases.

COMMENT.

In the light of the recent experimental and clinical evidence, a much discussed question again presents itself. Does posttonsillectomy abscess occur chiefly as a result of septic

embolism and infarction, or does aspiration play the chief rôle in its production?

The embolic theory rests in the main upon the postoperative condition of the tonsillectomy wound and upon the successful experimental production of lung abscess in the dog by the introduction of an infected artificial embolus. The posttonsillectomy wound situated in a septic field naturally contains thrombosed veins which are more or less infected. For the most part these veins are of small caliber, and very rarely is there any evidence of extension of the thrombotic process into the larger adjacent veins of the neck. If septic embolism from the tonsil bed were of frequent occurrence, one would expect numerous small emboli to be set free, with multiple secondary abscesses, not only in the lungs, but also in the course of the systemic circulation. In other words, one would expect a clinical picture of pyemia rather than a massive single lung abscess. Clinical and postmortem evidence of a generalized pyemia after tonsillectomy is very scant, the typical case being reported by Hedblom⁴ being one of the few found in the literature. The successful experimental production of a single lung abscess after the introduction of a large infected embolus into the jugular vein does not exactly reproduce the posttonsillectomy conditions. The experiment simulates rather closely the pathology embolism after sigmoid sinus thrombosis in which septic emboli are conveyed through the jugular vein, not only to the lungs, but also to distant parts of the body. The experiment undoubtedly explains most of the pulmonary complications in laparotomy performed under local anesthesia.

It cannot be denied that embolism from the tonsillectomy wound is possible, but in all probability it is of very infrequent occurrence.

The early experiments of Hoelscher⁸ and the recent work of Lemon¹⁰ show conclusively that buccal contents are usually inspired under general anesthesia, and were it not for the protective mechanism of the tracheobronchial tree lung abscess would be more frequently observed. The infecting agent is also important, as pointed out by Lord,¹⁹ who stated "that the incidence of postoperative pneumonia is five times greater in midwinter than in midsummer, probably due to the prevalence during the winter months of more virulent types of organisms

in the respiratory tract and their aspiration into the lung." It is difficult to explain the seasonal incidence on any other theory.

Bronchoscopy by different observers on a total of 400 patients has shown the presence of aspirated blood in from 40 to 78 per cent of all tonsillectomies performed under general anesthesia. Should this aspirated blood become infected and remain in the bronchi for a sufficient length of time, it seems reasonable to suppose that lung abscess might ensue.

I have demonstrated that aspiration of blood also occurs in tonsillectomy, performed under local anesthesia, in about 38 per cent of all cases. In these cases the quantity aspirated and the depth of penetration is usually less than that observed under general anesthesia. This would help to explain the greater incidence of lung abscess in tonsillectomy under general anesthesia.

The clinical experiments of Ochsner and Nesbit,¹⁸ as well as my modification of these experiments, show that under local anesthesia for tonsillectomy lipiodol may enter the smaller bronchi. The successful experimental production of lung abscess by the endobronchial introduction of pyorrheal material, or minced tonsil tissue as carried out by Smith,¹¹ Allen,¹³ and Crowe and Scarff,¹² would seem to remove all doubt as to the aspiratory origin of most posttonsillectomy lung abscesses.

CONCLUSIONS.

1. In tonsillectomy performed under general anesthesia, aspirated blood is found in the trachea and main bronchi in at least 40 per cent of all cases.
2. In tonsillectomy under local anesthesia some aspirated blood is found in the trachea in 38 per cent of the cases.
3. Posttonsillectomy abscess may be explained by the occasional prolonged retention in the bronchi of aspirated blood, crypt contents, tonsil tissue and buccal secretions.
4. The recent successful experimental production of lung abscess by Allen and Crowe and Scarff, show that retention of the infecting agent is an important factor in the causation of lung abscess.

5. Both clinical and experimental evidence indicate that aspiration of infective material is responsible for the majority of postoperative lung abscesses.

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707 RACE STREET.

XIX.

BLASTOMYCOSIS OF THE LARYNX.*

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When the clinical aspects of a presumably unusual disease become generally known and are correlated with the microscopic changes, the disease is sometimes found to be more common than was previously supposed. Blastomycosis of the larynx is probably such a disease; both clinically and microscopically it simulates tuberculosis and there are only three cases reported in the literature. Downing and Dennis, in 1918, reported the first case. Sartory, Petgès and Claoué, in 1923, reported the second, and Jackson, in 1926, reported the third and carefully reviewed all three cases. Two cases observed in the Mayo Clinic are reported here.

REPORT OF CASES.

Case 1.—A farmer, aged 51, came to the clinic May 31, 1919. Seventeen years previously the patient had been hoarse for six months following influenza and since then he had experienced slight shortness of breath. His chief complaint was hoarseness for the last year and a half. He had had influenza three weeks before examination; he had been slightly hoarse for a few days, then aphonia had developed. There had been some cough, particularly at night.

General examination showed nothing of significance other than hypertension; the systolic blood pressure was 180 and the diastolic 100. His usual weight was 180 and his present weight was 145, the loss having taken place in the last two years. Roentgenograms of the chest were negative. The Wassermann reaction and examination of sputum were negative. Examination of the larynx showed some scarring of both cords, a small

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glottis and a small web at the left vocal angle. There was a roughened area in the interarytenoid region and, possibly, ulceration. There was a small grayish nodule at about the middle third of the right vocal cord. Both cords were somewhat inflamed and thickened, especially at the vocal angle and in the arytenoid region (Fig. 1). Biopsy of the larynx showed inflammatory tissue only, with marked epithelial fibrosis. The patient was sent to hospital for observation and further study. On account of dyspnea it became necessary to open the trachea the next day. Dermatitis developed about the tracheotomy opening (Fig. 2). Microscopic examination showed the condition to be blastomycosis. Tissue from the larynx later showed the laryngeal lesion to be of the same nature.

The patient was given roentgen-ray treatment and potassium iodid internally. He returned January 8, 1920, at which time he had gained 40 pounds. Although the tracheotomy tube was still in place, his voice had improved a great deal. He returned again June 15, 1921. The laryngeal lesion was not active, but there was considerable scarring so that the glottis was reduced about half. September 9, 1922, still more improvement was manifest in his general condition. He had gained 12 pounds since the last examination, was working every day and had a good voice. Examination showed a cicatricial band extending across between the arytenoid, with limited movement (graded 2) of both cords. There was still some thickening of the right cord but apparently no activity of the lesion. The tracheotomy tube had been corked continuously for the last two years. The tube might have been removed at any time, but the patient was unable to stay to have it done.

The patient has not been seen since. However, follow-up letters show that he is still living and apparently well.

Case 2.—An ice man, aged 35, came to the clinic November 16, 1927, on account of hoarseness of seven months' duration. The onset of the trouble came with an acute cold, followed by gradual hoarseness and some pain on swallowing. There was no history of night sweats, cough or fever. His general health was excellent and he had gained seven pounds in the last six years. Tuberculosis had been repeatedly diagnosed and treated; a great deal of local treatment had also been given. Re-

peated biopsies, elsewhere, had revealed inflammatory tissue only.

The general examination was negative. Roentgenograms of the chest, examination of sputum and the Wassermann reaction were also negative. Examination of the larynx showed an extensive chronic process, which was inflamed and ulcerated, of both sides of the larynx, involving both vocal cords, the right false cord and the anterior portion of the left. It was rather mammillated in appearance, and some areas were covered with a thin grayish membrane. Both arytenoids were slightly swollen and there was some limitation of movement (Fig. 3). The first examination of tissue taken at biopsy revealed pathologic changes that resembled tuberculosis. On further examination, a diagnosis of blastomycosis was made. The patient was given potassium iodid, starting with 10 drops and increasing to 200 drops, three times a day. After being under observation for about a month, he returned home on account of illness in his family, with marked improvement in the laryngeal lesion and in general health. He returned a month later with the voice much improved. The vocal cords were thickened, but there was no demonstrable activity of the disease. His general health was excellent.

Comment: In these two cases, as in the three reported cases, the patients were males. It was impossible in either case to elicit definitely any history of exposure. In the first case, the history extended over seventeen years, although the hoarseness apparently cleared up between attacks. The shortness of breath during this time may have been the result of the laryngeal scarring. In the second case there was no obstruction, but the disease had been present for only seven months before treatment was instituted. In neither case was a similar lesion found elsewhere in the body.

DISCUSSION.

The final diagnosis of blastomycosis must be made microscopically (Fig. 4); nevertheless, a review of these cases presents certain unusual features that should suggest the disease, for example: a chronic, inflammatory, infiltrating lesion of the larynx with a negative history of syphilis, a negative Wassermann reaction, negative roentgenograms of the chest, and

negative examination of the sputum. A grayish, nodular infiltrating type of lesion, as if the mucous membrane had been touched with silver nitrate, was present in the three reported cases. The case reported by Jackson was also examined in the Mayo Clinic, but a microscopic examination did not reveal the blastomyces at that time. Later examination of the tissue corroborated the diagnosis. Broders, who examined the first specimen removed in Case 2, stated that the condition resembled tuberculosis, and this has been noted in other cases (Fig. 5). The blastomyces were demonstrated, however, when the microscopic field was darkened and carefully studied. He states that the multiple abscesses in the tissues make one suspicious of blastomycosis and that further study of these areas will reveal the organism (Figs. 6 and 7). Coccidiodal granuloma and tuberculosis must be ruled out in the differential diagnosis.

A careful microscopic examination in laryngeal cases thought to be primary laryngeal tuberculosis or chronic laryngitis of an indeterminate cause, may reveal the condition to be primary blastomycosis of the larynx.

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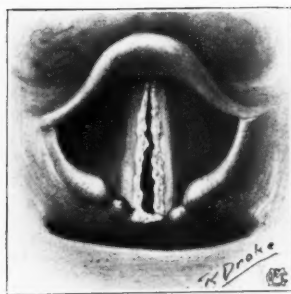


Fig. 1. Blastomycosis of the larynx. Both cords are involved; the small glottis is the result of scarring (Case 1).



Fig. 2. Involvement of the skin about the tracheotomy opening (Case 1).





Fig. 3. The grayish areas appear as though the mucous membrane had been touched with silver nitrate.

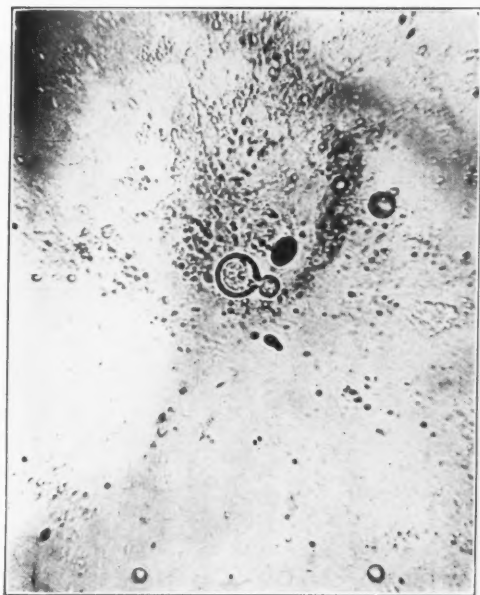


Fig. 4. Culture of blastomyces.





Fig. 5. (Case 2). A diagnosis was readily made from the biopsy specimen.

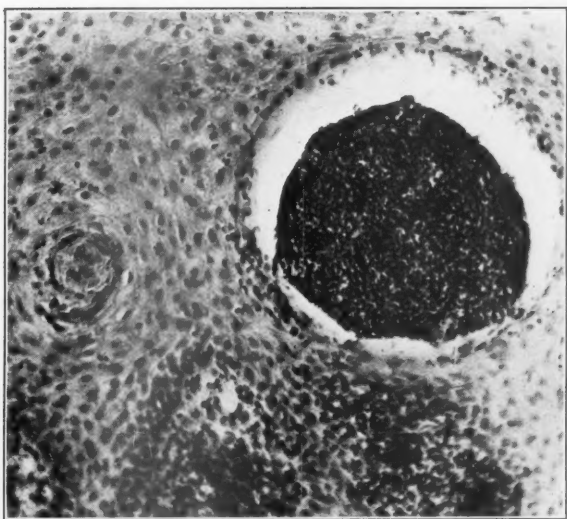


Fig. 6. Multiple abscesses suggestive of blastomycosis. A study of these areas with high power will show the organisms.



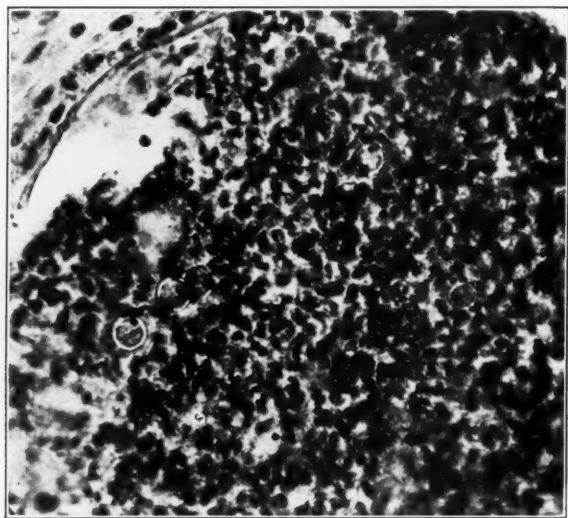


Fig. 7. Blastomyces seen in the center of the abscess.

XX.

VITAMINS AND DIETETICS IN RELATION TO OTOLARYNGOLOGY.

By BURT R. SHURLY, M. D.,

DETROIT.

What can this great medical profession and its highly specialized experts do to reach far underneath the problems of surgery, medicine and otolaryngology, and analyze our problems of infection and immunity, prevent the ravages of suppuration, of physical deformities and deficiencies and increase the span of life?

A world of work has been done, and many startling scientific facts have been brought to the light. If one-fifth of our profession are intimately interested in diseases of the eye, ear, nose and throat, if more than 70 per cent of all focal infection is in the tonsils, sinuses and teeth, and if the great percentage of 22,000,000 school children fall within the domain of our special work, is it not fitting and necessary that our knowledge and interest should be devoted extensively to a solution of the etiologic factors and the prevention of these alarming conditions?

It is, therefore, of eminent importance that otolaryngology throw its influence and scientific ardor into the realm of related biochemistry and the research problems so closely interwoven with our scientific progress.

The biochemic relation of lowered resistance to infection and temporary and increased loss of hearing in the presence of fatigue was demonstrated in my day at the University of Wisconsin under the direction of Professor C. F. Hodge. Many carrier pigeons were examined by cross section of the spinal cord after periods of long flight and after periods of resting and full nutrition. The protoplasmic granules of the giant cells were completely absent after flights and completely filled the cell after good nutrition and rest. It would appear as if the sal ammoniac in the battery was diminished and the spark was low.

While laboratory work has done perhaps more than any other branch of science toward clearing up many vexing questions about physiologic and pathologic activities, yet to be of lasting value and guidance it must agree with general and clinical observations. While this has been the case more often than otherwise, there are instances where laboratory observation and clinical data have crossed swords.

It is quite possible to realize the intimate relationship of infection in Waldeyer's ring and the lymphatic system, to remote and general systemic infection, when we perform the experiments of Cushing, who demonstrated by lumbar puncture and ventricular puncture at the same time that an indicator injected into the rectum could be detected in the cerebrospinal fluid in 45 seconds and finally in the ventricular dripping $1\frac{1}{2}$ minutes later.

The realm of dietetics in otolaryngology can be given no greater emphasis than in guarding the tonsil and adenoid child with marked lymphatism in its development, especially after operation. The experiments of Cramer, Drew and Mottram, showing the relation of vitamins A and B to lymphoid tissue are interesting to us. They found that absence of vitamin B from the diet of rats led to atrophy of lymphoid tissue throughout the body and also to lymphopenia. Polymorphonuclears were not affected. The absence of vitamin A did not tend to atrophy of lymphoid tissue and there was no leucopenia. Absence of B led to loss of weight, emaciation, abnormal temperature and what is termed marasmus. The condition can be produced by X-ray exposure and a destruction of the lymphocytes that play an important rôle in the nutrition.

My interest in the nutritional problems related to our specialty was greatly stimulated by a case of scurvy that entered the Detroit Eye, Ear, Nose and Throat Hospital. The case exhibited the factors of faulty diet, weakness, mental depression, hemorrhage from the gums, ears and nose, with a purpura and subperiosteal hemorrhage and tenderness. The diagnosis was confirmed by the series of blood counts and the rapid recovery with iron and calcium and vegetables. The anti-scorbutic vitamin water soluble C, found particularly in oranges, lemons, limes and tomatoes, with vitamin D, in red

bone marrow, with a tonic rich in calcium and containing malt, pulverized ossis, lactic acid, pancreatin and pepsin, was continued for some months. At the present time, two years later, the patient is well.

Raw milk is the most satisfactory single article of food taken by man. It supplies the deficiencies of grains, tubers and fleshy roots. It is rich in calcium and phosphorus. No other foods supply these essentials in quantity, except leafy vegetables. Serious damage to bone structure in the nose, face and elsewhere takes place unless the diet is properly constituted.

The experiments of Kaufman, Crickmur and Schultz assert that in rickets in rats there occur abnormalities of the osseous capsule of the internal ear which are identical with those changes in the long bones characterized in rickets and may interfere with the auditory function. Stepp and Fridenwald produced both rickets and xerophthalmia by a diet very low in vitamin A and phosphorus but rich in calcium. Nonrachitic infants and young children have between 10 and 11 mg. of calcium and about 5 mgm. of inorganic phosphorus per hundred grams of serum. All children under 2½ years with less than 3 mgm. of phosphorus had active rickets. Kramer, Casparis and Howland report the increase of phosphorus with the quartz mercury light is the equivalent of cod liver oil. Orr, Hotl, Willin and Borne believe that cod liver oil and the quartz light promote absorption of calcium and phosphorus from the intestine. Neither therapeutic aid, however, meets the defects in the composition of the diet, directly or indirectly, by supplying calcium or phosphorus, but by raising the potential of cellular activity and cell selection to influence ossification and calcification. Goldblatt and Scrammes observe with others that liver taken from irradiated rats is growth producing, whereas the livers from nonirradiated rats is inactive. The same was found true of lung and muscle tissue. It is demonstrated that experimental rickets may be produced in rats by an inadequate supply of vitamin D and disturbance of the calcium and phosphorus relation increasing with indigestion.

The daily problems of the otolaryngologist bring him intimately into the realm of the dentist and some of the pertinent questions of caries and pyorrhea, which seldom occur together.

Why do some persons escape? Is there a definite relation between diet and decay? For many reasons it was believed that caries was purely of bacterial origin. The propaganda of mouth hygiene has ignored some essential facts.

Faulty diet and tuberculosis have something in common. It is very prevalent in Labrador, where outdoor life prevails, with continuous ventilation but where a poor diet is the rule. It is essential that the protective foods, milk, leafy vegetables and fresh fruits, shall be taken in good quantity. This is important for the reason that the average American diet is deficient in calcium, over rich in phosphorus, often lacking in vitamin A, and in some C, as well as certain fat found with D.

One of the most interesting clinical studies in dietetics was made by McCollum in an institution of 234, with 84 children between the ages of 4 and 10 who were suffering from malnutrition. They were divided into groups and a quart of milk a day added to the regular diet. The results were phenomenal when added to the faulty and rickety diet selected from cereals, tubers, roots and meat.

Sound teeth, according to paleontology, existed before the toothbrush, but the great underlying principle remains within the domain of diet. In the expectant and nursing mother and the infant calcification begins between the fourth and fifth months of pregnancy, and the first permanent teeth in the last third of prenatal life. We watch over the ears, nose, throat and mouth of infants and children, and how keen is our interest to study with the pediatricist, or without him if not available, the dietary problems of the individual case?

Rypins, of Kansas City, examined 1,197 children between 3 and 6 years, and found 27.2 per cent had dental caries. Millanby, 1923, demonstrated that a substance like vitamin A has a potent influence in the calcification of the teeth. Puppies, fed during the period of development of the permanent teeth on deficient vitamin A, showed one or more of the following defects: 1. Thick and poorly calcified jaw bones and alveolar processes. 2. Irregularity in the arrangement of the teeth, especially the lower incisors. 3. Delayed eruptions of the permanent teeth. 4. Interference with the calcification of the denture. 6. Abnormal development of tissue at the gin-

gival margins and the peridental membrane. They continued these experiments in children with the questionable deduction that treatment has a very injurious effect on teeth.

The development of sound teeth with resistance is essentially a dietary problem. The small jaws and crowded teeth, with tonsils and adenoids, belong with faulty skeletal development and are influenced by dietary errors that favor rickets. Malposition also favors food packs and foci of fermentation.

Among the Italian children of the New York tenement district, between 1 and 8 years, one would seldom see a child who was not bowlegged. The teeth of Italian immigrant children arriving in Boston were far superior to those of like age raised in the United States.

Research in the field of nutrition applied to preventive otolaryngology offers an opportunity to raise the vitality of all mankind. The errors of diet may not cause deficiency disease, but insidiously their gradual operation, more or less constant, may be responsible for undermining the resistance in a degree so masked that the menace is quite unappreciated. As a cause of physical inferiority, instability of the central and vasomotor nervous system, lack of recuperative power and continued fatigue, loss of endocrine balance and lowered resistance, it stands out alone in the first rank.

I have used hypodermic iron citrate for many years in lowered resistance, poor appetite, adenitis and secondary anemia associated with focal infection. The hemoglobin will rise 5 per cent a week. This average was maintained in a large series of cases where daily doses were given for twenty days. The Italian preparation containing .01 citrate of iron, soda glycerophosphate, sodium arseniate and strychnin is of great value in selected cases. The tonic action on the vasomotor nerve centers gives the circulation in the glands and mucous membranes renewed vigor. In children, after tonsillectomy or in preparation for it, I introduce iron, phosphorus and calcium, by the use of a sandwich three times a day with meals, made by spreading bread and butter with saccharated iron and pulverized bone in quantity sufficient to cover a fifty cent piece. The child prepares this sandwich at meal time, and the importance of growing with properly selected food is given the valuable psychologic impression this important subject deserves.

In feeding rats, Evans and Bishop found that green leaves produce a profound effect on cell reproduction, because of vitamin E. The recent findings of McCollum that the function of vitamin E, in some curious manner, is associated with iron assimilation, confirms these clinical observations that I have made for years. A diet of liver with hypodermics of the green citrate of iron furnishes a combination rich in vitamin E, with increased value in assimilation and cell reproduction.

Eggs are deficient in calcium, high in vitamin A. It is estimated that six or seven eggs are equivalent to a quart of whole milk in vitamin B. Sea foods, such as oysters, add to the iodine and vitamin content.

In China and other parts of the Orient, where green leafy vegetables constitute a very important part of the food supply, rickets is almost unknown. Whereas, in Europe and America, where the tendency is to derive a large part of the food from milled cereals, meats, legume seeds and tubers, rickets and its frequent deformities in the nose and mouth are common.

In 1747, James Lind, a British naval surgeon, discovered that scurvy could be cured by the use of orange or lemon juice with the food. An interesting account by Dupain, 1920, details what happened in a large boys' school in England, during the winter term. Work and play fell far below normal. The boys became listless and irritable, and many minor complaints developed. Although they were well fed, nothing was supplied in uncooked foods with few greens. Fresh fruit was added to the dietary and the trouble ceased. Incipient scurvy from a lack of vitamin C was remedied.

While man requires vitamin C, rabbits, pigeons, ducks, geese, turkey and guinea fowl do not require it. Contact of food with copper utensils will destroy vitamin C, as reported by Hess in experiments with milk. Delf says that the juice of a medium sized orange or lemon or a fairly large tomato, daily, furnishes sufficient vitamin C for an adult.

It is becoming constantly more evident that even a slight departure from the optimal in the composition of the food may lead to states of nutritional instability which contribute to a physical breakdown when infectious processes are operating and the span of life may thus be shortened.

Larimore found that lesions in the esophageal portion of the stomach in rats occurred in vitamin B deficiency, consisting of an inflammatory edema of the mucosa and submucosa and localized ulceration. There was also an epithelial hyperplasia. In polyneurotic pigeons the adrenals increased two-thirds times in size, although the body weight and weight of the pancreas, thyroid, liver and stomach decreased.

Lecithin is shown to have a marked influence upon nutrition. There are doubtless many other valuable compounds in this unexplored field. The mystery is locked up in the accessory factors of diet. The importance of unraveling the unknown factors in ear, nose and throat pathology wrapped up in biochemistry may be appreciated only too well when we consider the high percentage that falls within our domain.

Terman stated that fourteen million out of twenty million had some physical defect and that two million had some grave defect of malnutrition; eleven million are said to have enough bad teeth to seriously impair health.

My experience with soldiers overseas and the reports from the draft would indicate that these statistics were not greatly exaggerated.

In a study of proteins, kidney, liver and milk stand out as of unusual value, and among the cereal grains wheat takes first rank, and a combination of two-thirds wheat and one-third protein gave the best results in growth. In discussing the question of high protein dietary and kidney damage, Newburg and Clarkson (1923) found that in most rabbits fed on 27 per cent or more meat they developed nephropathy with infection due probably to the excessive secretion of certain amino acids. These views are not supported by McCollum, who believes that a generous protein diet over a fair margin of safety produces the better growth and vigor in the young.

Chittenden's experiments reducing the accepted 116 to 120 gms. of protein a day to one-half are accepted by McCollum as advantageous. A fourth vitamin D is necessary.

If we are to have well nourished and resistant tissues in the ear, nose and throat, we must have a well balanced basal metabolism, as may be illustrated by the plus and minus findings in hyper- and hypo-thyroidism and the associated vasomotor phenomena. With the advent of elucidation in the field

of biochemistry, the importance of calories and inaccurate chemical analysis of foodstuffs recedes and the fascinating study of the vitamin jumps into the foreground.

The popular conception that any diet which complies with the composition of ordinary combinations affords and furnishes protein and energy in palatable and digestible form would promote growth and maintain health and long life is destined to pass into the background.

A ship of the Japanese navy, in 1884, made an eight months' cruise and many of the crew developed beri-beri. A second ship made the same cruise with an improved diet, and beri-beri was eliminated. It was not until 1897 that the scientific explanation was made, when beri-beri was produced in chickens in two to four weeks by feeding an exclusive diet of milk and rice.

It was, therefore, established that one of these was fat-soluble vitamin A, plentiful in butter and fat, egg yolk, cod liver oil, in the fats of the liver and kidney but almost absent in vegetable oils. The second, vitamin B, distributed liberally in natural food, such as tomatoes, soluble in water and dilute alcohol, but never found in animal or vegetable fats and oils.

Soubra found in cockerels fed without vitamin B that there was a decrease in the size and weight of the organs in the following order: Testes, spleen, heart, liver, kidney, pancreas and thyroid. Matthews reports the addition of vitamin B to the diet of infants suffering from metabolic disturbances increases appetite, weight and number of red blood cells. That resistance to infection is lowered is proven in the experiments of Guerrine, who fed pigeons on polished rice and found them no longer resistant to the bacillus of antrax. Rats and guinea pigs fed on a cereal grain showed delayed wound healing and lowered resistance to other infection.

In vitamin A deficiency, Blegvad reports a case of night blindness that failed to clear with six weeks of vitamin feeding. He made marked improvement in two to six subcutaneous injections of one cc. each of potent vitamin A solution.

Osborne and Mendel found calculi of calcium phosphate in the urinary tract of 91 animals in 857 autopsies, 43 per cent of which had a deficiency diet of vitamin A.

While drying of the lacrimal gland may be one of the first pathologic changes noted, the same effect takes place in the glands of the throat and mouth, and the animal may sneeze and cough violently. The mucous membrane of the larynx and trachea at autopsy shows xerotic changes. The salivary glands also are affected. In rats the secretion almost or entirely ceases. The cells on histologic examination are shrunk and irregular in size. The ducts of these glands show cornification and desquamation of the superficial cells. The sterility of rats is relieved by feeding lettuce, meat, whole wheat, wheat germ, especially the oil, rolled oats, dried alfalfa and quantities of milk fat. The vitamin X in this combination is classified as the fertility conferring factor. Lack of fat vitamin A or phosphorus leads to profound changes and loss of function in these tests.

Daniels, Armstrong and Hutton, in 1923, stated that rats in vitamin A deficiency may die of microbic invasion of the ear and nasal cavities before xerophthalmia appears. Feeding vitamin A does not restore the condition.

Hume shows that irradiation with a quartz mercury lamp prolonged the growth of rats without vitamin A, but failed to get response when they were on the diet over ninety days. Irradiation is not a substitute for vitamin A. In Michigan, and especially the northern peninsula, thyroid hyperplasia may be readily produced by diets deficient in iodine, and especially where animals live in dirty cages. His work also, in 1922, brought out the direct influence of infection in the mouth, nose, tonsils and gastrointestinal tract.

A detailed study of iodine deficiency in four counties of Michigan, where 31,612 children were examined, showed 14,914 had goiters, 40.5 per cent being boys. To remedy this, iodine was recommended in salt.

Rickets is prevented among young animals in the zoo by feeding liver, flat bones with marrow and fat and whole rabbits or pigeons. It is interesting to know that the disease is very rare in Greenland or Iceland, Norway or Sweden, or among the Eskimos. It is rare in the west of Ireland or Greece. It occurs more frequently between November and May. It is estimated by Hess and Unger that 50.75 per cent of artificially fed children in large cities have rickets; 96 per

cent of those dying under eighteen months showed evidence of the disease.

A presentation of these specimens of white rats prepared in our laboratory at the Detroit College of Medicine, and under the direction of Dr. Emmett, Parke, Davis & Co., confirms the findings of other observers, especially Daniels, at the University of Iowa, and demonstrates that diet deficient in vitamin A, which is plentiful in butter fat, egg yolk, cod liver oil and the fats of liver and kidney, exhibits on the 28th day of a lowered resistance to infection. It shows that these rats have a well developed nasal sinus infection and also extension to the middle ear. The controls in great numbers fail to develop suppurative. If the deficient diet is carried on later xerophthalmia results. The importance of these findings is illustrated by observations on children reported by Mari.

Mari, in Japan, describes 1,400 cases of deficiency in vitamin A, found among children of 2 to 5 years who were fed rice, cereals, beans, barley, vegetables. This was not found among fisherfolk and was cured with cod liver oil, chicken livers and eel fat.

Xerophthalmia, described by Livingstone, in 1857, after living on sugarless coffee, roots and meal, or by experiments on pure gluten and starch, shows the brunt of the symptoms in dryness of the conjunctiva and corneal ulceration extends to the lacrimal apparatus and the nose. Again, this pathology illustrates the inseparable relationship of the eye with the nose and throat.

Bloch reports a series of fifty cases of xerophthalmia in children fed separator skimmed milk, pasteurized and cooked again at home, together with oatmeal gruel and barley soup. These children developed borderline scurvy, beri-beri and ophthalmia in pronounced form. The cases certainly illustrate the unappreciated seriousness of malnutrition associated with tonsils and adenoids, and which in many cases fall but little short of a deficiency disease.

The association of night blindness, conjunctivitis and spring time catarrh was reported by Vetsch as relieved by cod liver oil. The relation of starvation for vitamin A to changes in the so-called glandular adipose tissue has been noted by Cramer. This variety of tissue is exhibited by the cretins in deposits

around the neck and axillæ and relieved by thyroid feeding. It is suggested that the glandular adipose formations are reservoirs of vitamin A.

The problems of hypothyroidism when exaggerated often show the following appearances in the nose, throat and ear: The nasal mucous membrane may be infiltrated and later thickened and the nose obstructed by a gelatinous waxy or yellowish secretion. The patient may complain of a full, cramped feeling in the throat and changes of coloring to a pale yellow or bluish tinge ensue. Slight edema with angioneurotic phenomena and a transparency of the interarytenoid fold without change of function in the vocal cords but with a dry throat or husky voice may be found.

It is recognized that the essentials in the prevention of rickets are vitamin D, with a well maintained ratio of calcium and phosphorus. One pint of cow's milk gives one grain of calcium oxid; human milk contains one-fifth as much. In children from 3 to 13 years of age, it was found that the optimal storage of calcium takes place when a child has a quart of milk a day.

The rôle of diet to increased susceptibility is well proven. The dietary of Voegtlin showed that the addition of liver was of great value. Manifestations of deficiencies in aggravated form, where a lack of phosphorus, iron, calcium are noted, may occur in the nose, throat and ear, as well as the general system. The secondary anemias, the lowered coagulation point, the poor skeletal growth and the thyroid imbalance call for a proper therapeutic selection.

In order that the hormones, or regulators of metabolism, may operate, it is essential that iodine shall be present to the extent required.

The observation of Goldberger in a Georgia asylum of adding fresh meat, eggs, milk and fresh vegetables in quantity, while controls were kept on the old diet, demonstrated that rickets appears in as early as thirty-four days to three months, but usually occurs between the sixth and twenty-fourth month. Late rickets may appear in older children or young adults. It is of particular interest to the otolaryngologist, because the most noticeable signs are seen in the bones, and those of the head especially. These children sleep poorly. They may

wear the hair from the back of the head rolling on the pillow. Constipation is the rule. The child does not walk. A rachitic rosary may develop where the bones of the ribs join the cartilages and pigeonbreast deformities begin. It is probable that the otolaryngologist sees more of these onsetting symptoms with tonsils and adenoids than any other special worker except the pediatricist. The deformities of the nose, nasopharynx and hard palate, the square shaped head, the bony formations on the side and front of the skull are developed. Curvatures take place and the jaws and epiphyses swing out of alignment.

In conclusion, it may be said that while the research laboratory alone will be able to furnish a final solution to the interesting relationship that exists between food and pathology in the ear, nose and throat, and elsewhere, we may well believe that a genuine practical application of dietetics and therapeutics is indicated in otolaryngology together with our surgery. The demand of the human economy for iron, phosphorus, calcium, iodine and vitamins is present in otolaryngologic pathology more frequently than in other deficient parts of the body. Our rôle and our influence in the realm of pediatric otolaryngology is most extensive, as malnutrition, adenitis and infection are more prevalent in our domain. If, therefore, I have succeeded in attracting your interest and study in this direction my mission will be fulfilled.

XXI.

THE MANAGEMENT OF MALIGNANCIES IN OTOLARYNGOLOGY.*

BY JOSEPH C. BECK, M. D.,

CHICAGO.

In accordance with a previously expressed promise to make a follow-up report at any time that something new could be added to our work on malignant disease, we wish very briefly and concisely to place on record some experiences that should prove to be of definitely practical value, especially from the standpoint of therapeutics. Not wishing to be too hasty in making this report, we have allowed more than five years to elapse to enable us to state definitely that the methods to be described have been curative in a sufficient number of cases to give a little more encouragement to patients afflicted with cancer.

The experiences mentioned above were with X-ray, radium, both combined, surgical diathermia, laryngectomy, radical resection of the superior maxilla and *en bloc* gland resection of the neck. None of these measures are new to the majority of medical men, yet the application of them and the observations following their use involve distinct personal equations and therefore our report is made at this time.

After a most discouraging period¹ of operating and treating malignant disease, which was usually discovered or diagnosed late, and the subsequent irradiation by X-ray and radium as well as by the aid of surgical diathermy, we have now developed an atmosphere of confidence that borders on over-enthusiasm, with the result that we fear a setback. Why are these better results obtained? In the first place, much earlier diagnoses, with the aid of biopsy, are brought to us, especially in the case of malignancies of the larynx and tongue. Second, we have been absolutely "sold," so to speak, on the operation of

*Presented before the Detroit Otolaryngologic Society, December 21, 1927, and the Chicago Laryngologic Society, January 9, 1928.

laryngectomy, even in cases of glandular metastases in the neck. Third, radium needling and seeding under the supervision of an expert radiologist. Fourth, the use of deep X-ray therapy, also under the control of an expert radiologist. Fifth, surgical diathermia by means of the most advanced instrumentarium. Sixth, the surgical technic of operating on the glands of the neck in their removal *en bloc*. Seventh, the subsequent plastic surgery and prostheses to make up for defects produced either by operation or disease, are additional factors in the happier results obtained. Eighth, the general physical examination and the care of the patient before and after operation—in other words, the cooperation of an internist, is of inestimable value in this work. For example, the knowledge that the patient to be treated or operated upon for malignancy is also a diabetic, make for a better result, since the patient is properly prepared and treated by insulin, and can stand the operation, whereas before the advent of this wonderful remedy, insulin, the patient was taking a great risk or was an inoperable case.

The conditions about the head and neck that we have treated in this what might be termed "newer attitude" are as follows:

A. Carcinoma.

B. Sarcoma.

Carcinoma:

1. About the lids.
2. Lids and face.
3. External nose.
4. External nose and upper lip.
5. External ear (pinna).
6. Pinna and auditory canal.
7. Alveolar process of the superior maxilla.
8. Alveolar process and antrum of Highmore.
9. Septum nasi.
10. Inferior turbinate.
11. Velum palati.
12. Tonsil.
13. Tonsil, plica and base of tongue.
14. Anterior two-thirds of tongue (central and marginal).
15. Inner surface of cheek (region of ampulla of Steno's duct).

16. In a leukoplakia of cheek.
17. Parotid gland.
18. Submaxillary salivary gland.
19. Lower jaw.
20. Larynx in the various portions and positions, including epiglottis and base of tongue.

Many of these cases had involvement of the lymphatic glands of the neck. In all of our cases a biopsy was insisted upon preliminary to the operation, and great care was exercised in that procedure, both in clean cutting methods and in going deep enough to feel assured that a real portion of the growth was removed for diagnosis. Frozen sections were usually made after a period of hardening in formalin for twenty-four hours, but paraffin sections were also ordered when very small particles were examined. The pathologist's report on the differentiation of cancer cells is always considered of the utmost importance in the prognosis and the anticipation of recurrence, as, for instance, a basal cell carcinoma or a prickle cell carcinoma is certainly less malignant than squamous cell, which, in turn, is more malignant than the scirrhous type or even an adenocarcinoma. In several cases we encountered a pigmentary form of cancer, and this type was considered and found to be most malignant. In other words, the cooperation of the pathologist has been of inestimable value to us. May I add that every section was studied personally by us after the pathologist made his diagnosis. This, we feel, is of special benefit to the clinician because he much better appreciates the activities that are entered into in each individual case.

In sarcoma, a more helpful attitude does exist, owing to the fact that when the biopsy shows a greater preponderance of small round cells very clearly differentiated with considerable fibrous tissue and not too many blood lakes, the treatment very many times proves to be permanently curative. We have a case (temporary resection of the upper jaw) of cure of twenty-five years' standing, wherein straight surgery was resorted to. We also have quite a number of cases wherein cure is of a briefer period of standing, in which surgery plus X-ray, and particularly radium, was employed. In those cases where the biopsy showed a lesser differentiation of cells, say mixed cell sarcoma with large blood lakes and very little stroma,

especially if pigmentation granules appeared in the section, recurrence was invariable and rapid death resulted. The cases of lymphosarcoma or osteosarcoma proved to be hopeless, although many of the patients appeared to respond very well to the X-ray and particularly the radium treatment, but only to recur with ferociousness and a resultant rapid death of the patient.

In regard to the technic of the straight surgery, I desire to call attention to certain advances made in recent times that have a great deal to do with the expectancy of an increased number of cures from malignancies. Many years ago Wertheim of Vienna showed a large series of cases of carcinoma of the uterus in which he performed, in conjunction with a complete hysterectomy and adnexa removal, the radical dissection of the pelvic glands. Many of these cases remained without recurrences. The same principle was tried and found to be true in operations of the oral, pharyngeal and laryngeal cavities wherein the glands of the neck are removed radically, usually "*en bloc*."² Not only are the glands removed, but contiguous tissues, and even the resection of the internal jugular veins is done. The carotid arteries are frequently exposed or lying bare in the entire length of the neck in danger of infection and developing an arteritis with subsequent dilatation and aneurysmal formation. I had such an unfortunate occurrence in the case of a physician in whom I was compelled to do a sudden ligation of the common carotid artery with the terrible complication of hemiplegia.

Experiences such as these have led us to advocate ligation of the external carotid before the attack is made upon the malignant growth. This causes us to perform the operation upon the neck first. Occasionally upon opening the neck wide, we find the common carotid enveloped in a network of malignantly infiltrated glands and lymphatics, in view of which resection of the entire mass is indicated. The pneumogastric nerve is carefully isolated and dissected free and a Crile clamp applied to the common carotid, after which the skin may be loosely united. The clamp is tightened daily, and thus allows the establishment of a collateral circulation, preventing such complications as death due to cerebral anemia and edema, or hemiplegia consequent to the subsequent encephalomalacia. The

neck is reentered at a future period after the establishment of a competent collateral circulation and the radical excision, including glands, jugular, common carotid, and muscle effected.

In the cases of carcinoma of the hard palate and antrum, base of tongue, floor of the mouth, soft palate and cheek, the coagulation must be carried frequently into the neck, and consequently the carotid artery, especially the external, is endangered by possible scorching with subsequent weakening of the wall and possible aneurysmal dilatation and secondary fatal hemorrhage. To prevent this we ligate the external carotid and do a gradual compression ligation of the common carotid if necessary. Following these "en block" gland removals, the wounds are left wide open for observation of recurrences as well as the irradiation with radium and X-ray.³ Not having to go through the skin, the radiologist can apply the large amount of the short rays which should take care of any recurrence, if any take place. The covering of the skin defect can be performed at any time after one is certain of the cure.

In regard to the laryngectomy, we are all indebted to Mackenty for his development of a most perfect technic of the old master Glück's procedure. When more men have learned to perform this operation there will be many more lives saved. As is the case today, many carcinomas of the larynx are subjected to X-ray or radium treatment rather than to operation, and most of those, if not all, succumb to the disease.

The question of laryngofissure, window resection and hemilaryngectomy for carcinoma of the larynx is still a debatable question. Especially is this true now when surgical diathermia is employed. Personally, I have had some very happy results in well selected cases—that is, of small neoplasms on the anterior part of the vocal cords, wherein no glands in the neck were present. I have also, in former years, operated upon a too large number of carcinomas of the larynx by a lesser procedure than laryngectomy followed by X-ray and radium. In these cases recurrence and death followed. Had I performed laryngectomies in these cases with gland dissection and the removal of any other suspicious tissue in the neck, such as the upper part of the esophagus, base of the tongue and muscles, some of these cases could have lived. I know that some will question the advisability of living after such an operation and

that should be left to the individual to decide. It is the function of the voice that is uppermost in the mind of the patient, and the contemplation of going on the rest of one's life without speech is very depressing. However, if one takes the time to explain that by aid of training, an esophagobuccal voice is very serviceable and that great progress is being made with the artificial larynx, the sad outlook is decidedly mitigated.

Technic of Surgical Diathermia.—The patient is prepared as for any other type of major surgical procedure. The anesthesia is varied to suit the individual case. Where simple coagulation of the growth is all that is to be performed, the region is rendered anesthetic by either nerve block or infiltration anesthesia. When a more extensive operation is performed the patient is given the benefits of synergistic analgesia, which is a modified rectal anesthesia used in conjunction with subcutaneous injections of morphin and magnesium sulphate. Occasionally, patients are somewhat refractory to this anesthetic and it will require supplementary anesthesia with interrupted chloroform inhalation. Ether, for obvious reasons, is to be shunned.

Incision.—We make the incision with the so-called radio knife. Our machine has various modalities for varying degrees of electrocoagulation and desiccation. The high desiccating current is used for the incision. After the proper adjustments, the instrument is tried out on a piece of raw meat before operation. The knife should be so adjusted that the skin tissues seem to fall apart in the way of the advancing knife. No bleeding is encountered unless a larger vessel is severed. This may be closed by direct sparking to the bleeding point or it may be caught with a hemostat and the current applied to the hemostat, direct, and even a ligature may be necessary. One should be very careful as to the manner in which the indifferent electrode is attached to the patient's back. It must lie in very firm contact and the spot should be moistened with soapy water. Adhesive and bandages should be applied to make the contact firm. Poor contact or a slipping, indifferent electrode will be certain to produce a severe burn to that region, and such an accident would prove embarrassing to the surgeon were he asked for an explanation. The electrical constants vary in the type of machine used. In the instrument employed by us, the

autotransformer is set at about 10 and the lowest voltage terminal is used. The spark gap is adjusted to read about 1200 milliamperes. These figures may be varied to suit the individual case.

Coagulation.—When the incision has been performed and the growth isolated for the actual coagulation, or “cooking,” the active electrode is changed from the knife type to the needle type. We prefer the Planke needles. These are made in various lengths and also fit the handle which holds the knife loop. The medium or high voltage terminal is used, depending upon the depth of coagulation required, and the autotransformer set at 6 or 7. The spark gap is opened to read between 1,200 and 1,400 milliamperes. The needle is plunged into the neoplasm and the current turned on until an area of coagulation occurs about the needle. The current is then closed and the needle plunged into the adjacent tissue and the performance repeated. When a large part of the tumor has been treated in the manner described, the resulting coagulum is removed by scraping with a curette until moist and bleeding tissue is reached, when the coagulation is resumed. This is continued and the entire tumor destroyed. We must be sure to remove the entire growth, and even go into the healthy tissue. Bleeding is frequently encountered and is stopped in the manner above described. When the entire neoplasm is destroyed we prefer to coagulate the base with the Holmgren electrode or button. In the coagulation of the cavity of the antrum, Holmgren¹ recommends the use of two similar electrodes, the active and inactive, in close proximity to one another within the cavity. The reason for this procedure is to avoid the passage of all the current through the mass of tissue from the back to the antrum, thus obtaining a more efficient action.

The reaction from this type of treatment is surprisingly mild and the systemic reaction is practically nil. The reasons for this are explained by various writers differently. My own belief is that there is very little bleeding, and, consequently, not much chance for retentions, infections and resorption. The cut surfaces are surely sterilized by the heat. The lymphatics are undoubtedly sealed by coagulation. The after-treatment varies in each individual case; however, certain generalities can be made. It always takes a long period for the wounds to

cleanse themselves. The exudate should not be removed mechanically. The odor is rarely of the putrid character, as it is from actual galvanic cautery or from a regular surgical operation in and about the cavities of the nose, mouth and throat. Should putrefaction or bad odor be present, balsam of Peru or mercurochrome will take care of this. Following surgical diathermia about the bony cavities, there is always considerable sequestration expected. Whenever a loose piece of bone is found, it should be removed because healing cannot take place until all the dead bone is removed. Excessive granulations are kept down by surgical diathermia or by the aid of silver nitrate. It is well to remove small bits of the granulations during the healing process and send these to the laboratory for the checking up of possible recurrences.

X-ray Socalled Deep Therapy.—This we have always left to the radiologist. We keep the patient under observation with control examinations of the parts involved. Suffice it to say that the patient is kept at the hospital during and a day or two after a socalled series. The proper dosage is calculated by the radiologist, three days being usually allowed for the series, with two exposures daily. The patient is given an appointment to return for a similar treatment within two or three weeks, depending on the type of growth and its location, also as to what progress has been made. The socalled radiotoxemia, as well as the local reactions, particularly the edemas and cellulites, are often alarming. The effect on the salivary secretion (extreme dryness) is frequently complained of. The further elaboration of this toxemia will be taken up in a subsequent publication on some of the research work I am doing with Togggenberg Swiss goats.

Radium.—This is used in forms of placquest, capsules, needles and emanation seeds. As in the case of X-ray, the dosages of radium are figured out by the radiologist (shall I rather say the radiumologist?) depending upon the type of growth and its location. Whether the plaques, needles or seeds are to be used, this must also be determined jointly by the clinician and radiumologist. The applications, at least in the case of the otolaryngologist, are to be done by himself, as he is better prepared to handle them here. One does not expect any immediate traumatic effect from the use of plaques or

capsules. However, in the case of the needles and seeds, very definite traumatic lesions must be reckoned with. In the case of radium emanations in seeds, one must figure on the life or period of activity of the emanations. The dosage of the emanation seeds is determined by the radiologist, and they are prepared with an overdosage so as to allow for the decrease in activity from the time of preparation until they are implanted into the neoplasm. The implantation is performed by the laryngologist, and the seeds are allowed to remain indefinitely. They may, however, be removed after the expiration of their activity, which, as a rule, takes from five to ten days. In rare instances they have to be removed by reason of their location, as they may fall into the larynx and trachea and thus cause complication.

Surgical Technic.—We in reality perform a two-stage operation, first upon the neck and subsequently upon the primary growth. If the condition of the patient will permit, the entire procedure is performed at one time. The neck operation is performed first in order to ligate the external carotid, which acts as a preventive of aneurysmal dilatation of the vessels or hemorrhage following the radical procedure on the primary growth. This is performed in every case, as we are enabled at the same time to explore the neck for metastasis and many times have been able to demonstrate malignant infiltration in the neck, even though glands were not palpable before operation.

A skin flap is outlined and dissected free. The sternomastoid is isolated and pulled to one side, exposing the underlying carotid sheath, which is incised and entered. The vagus is carefully isolated from the vessels and a temporary tape ligature is passed around the common carotid at the base of the neck and left untied. This is an important precautionary measure, because we can instantly tie the ligature if confronted by an uncontrollable hemorrhage in our work higher up. The external carotid is next exposed at its origin at the level of the thyroid cartilage, bearing in mind that in this location the external carotid is internal to the internal carotid, and a double ligature is passed around it and doubly tied. Any and all suspicious glands found during this procedure are resected, and frequently the jugular will be found to have been incor-

porated in a mass of carcinomatous glands and lymphatics. If this be the case, the vein is ligated as high up and as low down as possible and resected with the glands "en block" between the two ligatures. In those instances where the common carotid is involved, a Crile clamp is applied for a number of days until a satisfactory collateral circulation has been established, when the neck can be reentered and the common carotid resected with the malignant mass after a secure ligature has been applied. In all of these procedures the integrity of the vagus must be respected. Accidental ligature and even resection of the vagus has occurred, and this is especially prone to occur in those cases in which the neurovascular sheath is so matted together by the neoplastic tissue as to entirely disorganize the anatomic picture. If this unfortunate event occurs, respiratory inhibition and syncope may result. The entire neck is thus freed of all grossly malignant and suspicious tissue, even, if necessary, following the chains of glands and lymphatics into the floor of the mouth or into the posterior triangle of the neck. A judicious application of electrocoagulation is now applied to the exposed surface and the wound should be left open for future radiotherapy.

Laryngectomy.—We have definitely concluded that except in specially selected cases, anything less than total laryngectomy, when confronted by a laryngeal malignancy, is only a temporizing measure and is to be condemned. We follow closely the technic of Glück as modified by Mackenty, which needs no elaboration here. Surgical diathermia is not used.

Carcinoma of the Lip.—A radical, wide excision of the growth is performed with the radioknife, the contributory glands being resected as above described, and the neck wound left open.

Carcinoma of the Tongue.—A hemisection of the tongue is performed by means of the radioknife and electrocoagulation. The preliminary ligation of the vessels is highly essential to prevent dangerous hemorrhage.

Carcinoma of the Tonsil.—The growth and surrounding tissues are widely destroyed by electrocoagulation. The external carotid should have a preliminary ligation in all cases.

Carcinoma of the Palate.—Radical wide destruction of the growth and surrounding structures with electrocoagulation is

effected. If the disease extends beyond the midline, ligation of the palatal vessels on the opposite side may be necessary.

Carcinoma and Sarcoma of the Antrum.—The skin is incised and dissected with the radioknife and the growth removed by electrocoagulation as wide as possible. We are in accord with the views of Schmiegelow⁵ and Holmgren and Berven,⁶ that the extent of the operation should not be influenced in the least by cosmetic considerations, and, if necessary, the skin of the face, the entire maxilla, the orbit and the eye should all be sacrificed. The consequent deformity can be remedied at a much later time, months after a presumptive cure has been established, by a plastic operation or prosthesis.

The postoperative observation of the wound is of the utmost importance, and a careful watch must be kept, all frank recurrences and suspicious granulations are destroyed by electrocoagulation and occasional checks upon the granulations should be made by biopsy.

Irradiation with X-ray and radium to the open wound may be given at the discretion of the operator.

Attention may be called at this time to the prevention of pain in hopeless cases. Morphin, not infrequently, is of no aid, especially when administered over long periods of time and when tolerance to the drug has developed. We are depending upon alcohol injections into the nerves at their emergence at the base of the skull and also root resection of the gasserian ganglion for the control of pain in these obstinate conditions. It might be mentioned that at present the use of inhalations of trichlorethylene is receiving a great deal of attention in the control of pain of the trigeminal area, but as we have used it for only a short period of time we are as yet unable to definitely evaluate its efficacy.

CONCLUSIONS.

1. In surgical diathermia we have a destructive surgical agent which is decidedly lethal in its action upon malignancy.
2. Until the specific etiologic factor in the production of malignancy is established, the cure of cancer is dependent upon radical surgery and irradiation.
3. A sufficient number of cases of cancer cured by surgery and irradiation as outlined above, are on record to encourage

us in the continuance of the management of these cases in this manner.

4. The cooperation of the various specialties are indispensable in obtaining satisfactory results.

5. Prevention of latent hemorrhage following surgical diathermia is effected by prophylactic ligation of the external carotid and gradual compression ligation of the common carotid.

6. Pain before, during and after operation may be best controlled by nerve block with alcohol or even gasserian ganglion root resection.

7. Statistics are only of value when the reporter and his results are under the supervision of an association such as, for instance, the American Society for Control of Cancer, because statistics as now gathered are of distinct personal equation.

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XXII.

OTORHINOLOGIC PATHOLOGY OF SWIMMING.*

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Social phenomena invite the attention of scientific medicine when rapid changes occur in the habits of considerable numbers of the population. The excellent ideas of life saving organizations, of physical trainers, and of recreation and child training groups have been embodied in a very great increase in the number of public bathing places. Swimming is taught by every athletic club, community house and welfare association that can afford a pool. A pretext is offered to fashion and a temptation is held out to the libertarian ideas of youth. Vanity or the crowd impulse drives many persons to the water who are lacking in physical stamina or are characterized by defects which render them vulnerable to water borne pathology.

We may recall the remarkable development of public bathing in ancient Rome—without clothing, and followed by oil friction of the skin—and may remember that a certain degree of individual privacy in the sterner days of the early republic was succeeded by vast public establishments permitting mingling of the sexes.¹ Various contagions were ascribed to these gathering places, and not least was the moral hazard involved. For this reason, upon the rise of the Christian power (and by reason also of the decay of the system of aqueducts during the various barbarian sieges of Rome), public bathing places received the bad repute of their Roman originators and were not reestablished until the middle ages.²

A similar cycle of personal privacy and innocent modesty succeeded by mixed bathing in public places occurred in the medieval towns of western Europe.³ Possibly a taste for bathing was revived on the return of various Crusaders from cities where the Saracens maintained bathing establishments.

*Read, by invitation, before the New England Laryngological and Otological Society, Boston, January 9, 1928.)

It seems probable that the great plagues of the fourteenth and fifteenth centuries were spread from public bath houses, as well as by more usually considered methods of respiratory contagion. Such places were closed by the authorities during epidemics.⁴ The term "bagnio," which has come down to our day, indicates the low moral estate which finally drove the public away from these resorts.⁵ Fear of respiratory contagion, amusingly caricatured in the huge false noses full of aromatics worn by medieval doctors in attendance on plague cases, was sufficient to scare many people from any sort of bathing—except at birth and death—for nearly two centuries. Hostility of physicians of the Galenic school to the barber surgeons who worked in bath houses is said to have caused many obedient patients to discontinue bathing except at home, and then only upon the doctor's prescription.⁶

It will be noted that some of our problems, in the field of respiratory and of moral pathology, have cropped up before in the history of civilization.

Aquatic animals seem to be more fortunate, to judge from the researches of Ingersoll⁷ and more recently of Marshall Taylor.⁸ And many species of mammals, notably those most frequently used for laboratory work, maintain a high degree of immunity to water irritation and to infection through the nose and ear thus introduced.⁹ Experimental infection with highly virulent cultures from accessory sinus disease could not be effected by us in a series of rabbits, by introducing the liquid into the nostrils; nor was their apparent immunity lowered by chilling the body in a subsequent experiment. Two factors are probably responsible: the relatively high body temperature of these animals, and the relatively small size of any accessory nasal cavities. Instead, a very vascular and much foliated ethmoturbinal structure takes care of the olfactory terminals. Experiment upon the higher monkeys should be used if accurate findings are desired.⁴³

Flaps, sphincters and valves which exclude water from nose and ears in purely aquatic species are almost equaled by voluntary musculature in land animals which swim occasionally. Puckering or twisting the nostril or auditory meatus for such protection survives in man, unhappily, only as a vestigial function. The rhinologist is reminded of it occasionally during

examination of an unruly patient. Other methods of defense must, therefore, be used by man if water is to be excluded.

External factors in the pathogenesis of conditions secondary to swimming may be most important. Loss of body heat, especially by slender persons, rapidly lowers resistance. Immersion in very cold water over a long period may not prove harmful to a robust man who is actively using his muscles. Let him keep quiet, and a very short exposure may prove serious. Along a windy beach, chilling is worse than by indoor pools, because of rapid heat loss from evaporation in the open air. Length of time during which the body remains damp and cool is a contributory factor. Taylor's exhaustive researches showed lowering of body temperature half a degree an hour when children were standing about in wet bathing suits.^{8 10} Hygienically, nude swimming is far less dangerous than wearing any sort of bathing suit to hold water and absorb heat. Cotton and silk-cotton mixtures are especial abominations in this regard. Light wool is best in spite of its weight when saturated. "Swimmin' hole" experience in boyhood bears out the recent practice of professional distance swimmers in the matter of freedom from clothing. Long immersion, of course, calls for oiling of the skin to prevent softening of the surface layers and to insulate somewhat against heat loss.

Salt water is popularly considered more healthful than fresh, and this is borne out by statistics of the higher incidence of disease from fresh water than from salt; and from salt ponds or tanks rather than the surf.^{10 11 12} We must not forget, however, that taste is an effective warning to the swimmer to keep salt water out of his mouth and nose; he makes no such effort with water which is apparently fit to drink. Probably this involuntary impulse toward self protection may account for such statistics, rather than any fancied superiority in germicidal power. Also, the saline content of such water renders it hypertonic and hence less destructive to the cell contents of the skin and mucosal surfaces than is plain water.

Chemical antiseptics—especially chlorin—may seem slightly irritating, a factor that may be gauged by the degree of conjunctival reddening. As with chlorin gas, persons with any idiosyncrasy will have serious discomfort from turbinal swelling. This is, however, the cheapest and easiest method of

disinfecting large pools, especially when the water cannot be changed very often. A considerable amount of solid matter accumulates in tanks which do not operate under constant flow conditions, but this can be skimmed off, and certainly sterilized if it does not float, by chlorination. An oily scum will sometimes accumulate in a tank during the quiet hours of the day between gymnasium groups, and with constant flow from one end to the other will become concentrated near the outlet end.^{13 14 15 16} The first bathers to enter after such a rest period may receive a massive dose of such an accumulation, which is later scattered by the agitation of the water during swimming and diving. Through the cooperation of the Oregon State Board of Health, Dr. Wm. Levin has furnished me with the following tabulation of conditions in all the indoor pools of Portland, Ore., from samples taken December 8, 1927:¹⁶

Tank	Bacterial count per cc.	B. coli per 100 cc.	Residual chlorin PPM	Reaction	Chlorides PPM	Total solids PPM	Tank Last Emptied
Couch School	7	0	0.2	alkalin	73.7	610	4½ mos.
Multnomah Club.....	210,000	100+	.0	acid	2.9	39	4 days
Portland Natatorium.....	300	0	.0	acid	13.6	22	2¼ mos.
Y. W. C. A.....	12,700	0	0.02	acid	2.9	24	4 days
Y. M. C. A.....	2	0	0.05	alkalin	32.0	78	2¼ mos.
Elks Club	2	0	0.3	acid	9.7	170	3¼ mos.
Neighborhood House.....	5	0	0.05	alkalin	24.2	112	4¼ mos.
Buckman School	6	0	0.03	acid	21.8	87	1 mo.
Y. M. C. A. (Northeast)	1	0	0.15	acid	25.2	47	3 mos.
K. of C.....	7,500	100	.0	acid	2.9	22	10 days
Turnverein Hall	8	0	not made	acid	2.9	36	4 days
B'nai Brith	27,400	0	.0	acid	2.9	24	4 days
Shattuck School	3	0	0.15	alkalin	88.9	684	3½ mos.

It is interesting to note that pools which use chlorin disinfection in this list have a very low bacterial count, but run high in solids and chlorids. The impression among otolaryngologists in Portland rather accurately ascribes to the high bacterial count in certain places most of the swimming pool pathology seen by us. Occasional exceptions, sometimes of fulminating virulence, may be due to massive doses of solids containing a few germs—surface contamination as outlined above.

Most important, perhaps, among extrinsic factors in pathogenesis is the style of swimming. With the old breast stroke, the head does not need to be under water; but with the various crawls, overhand, speed strokes and back strokes, partial immersion of the head and face becomes essential. Recall that these strokes have come up to universal popularity only within the past ten or twelve years. It is not surprising that many beginners acquire faulty breathing habits, inhaling a little water into the nostrils each time the face rolls under. The nose should always be used for exhalation, the mouth for inhalation, when indulging in crawl swimming. Many good teachers start the pupil floating face down in the water, allowing the air to bubble out of his nose. This is an excellent and safe way to learn and will do much to obviate complications from faulty breathing habits.

Even more important is proper breathing during the dive. Some of the worst cases in our clinical literature are those of unskilled persons who jumped in "feet first" and received a "nose full" of water. If sufficient breath is not secured before the headfirst dive, and if slow nasal exhalation be forgotten, it is readily possible for the hydrostatic pressure of eight or ten feet of water to invade not only the nasal airways but the maxillary antra, frontals and even the sphenoids. The squeaking or bubbling of escaping air is a phenomenon mentioned by many divers preceding a so-called "cold in the head."¹²

Except for matters of faulty engineering technic in tank purification or antiseptic treatment, or of massive contamination by a very great overload of bathers with inadequate water change, the hygienists and public health authorities are keeping our pools reasonably clean.^{13 14 15 16 17} Epidemic transmission of certain types of hemolytic staphylococci concerned in furunculosis has been repeatedly observed. Following "flu"

epidemics, when attenuated strains of respiratory streptococci are present in a large percentage of the population, it seems probable that numerous accessory sinus and otitis cases may develop among patrons of swimming pools. Severe epidemics of this kind are rare when modern methods of tank management are in use. Colon bacillus infections of the antrum, usually ascribed to bad molar teeth, are not uncommon, and may well have their origin from contaminated water used in swimming.

The introduction of abrasives, such as sand and saline crusts, facilitates invasion of the skin of the nasal vestibule and auditory meatus by bacterial agents as well as various yeasts and fungi.¹⁸

Turning to consideration of the intrinsic factors in the pathology of swimming, we are immediately faced by the fact that relatively few out of each large group using a given pool, bathing beach or river resort succumb to otorhinologic disease. What are the personal factors rendering these individuals vulnerable, while the rest are strengthened and refreshed?

The adipose insulation of well nourished persons keeps them from chilling so rapidly as thin people. On an average, therefore, women—unless they are actively engaged in a starvation attempt to reduce weight and are thus lowered in resistance—can remain in water or wet garments longer than men without harmful effects. Small boned individuals, having less weight to sustain in the water, tire less rapidly and are therefore less likely to exceed the limits of muscular endurance. People with long, narrow thoracic cavities, unaccustomed to full use of the lung apices, are more likely to make errors in the technic of breathing while in the water; also their index of flotation is below that of the barrel chested, chunky, deep breathing type. Such matters of physique, largely hereditary, should direct the special attention of physicians and swimming instructors to children and young adults thus handicapped, and ironclad rules requiring all students in a given institution to learn swimming should be relaxed in such cases.

Locally, any narrowing or distortion of the nasal chambers or auditory meatus favors retention of water and delay in its removal. Septal deflections, spurs and thickenings, collapsed

alæ, large turbinates, adenoids, polyps—any and all obstructions to breathing—are, more or less, predisposing factors toward nasal pathology from swimming. So also with the narrow auditory meatus, especially if greatly bent in either curve, if heavily beset with hairs or viscid cerumen, or if encroached upon by osteomata.

Acute infectious processes, and especially those chronic infectious disorders to which the individual has become somewhat calloused—alar or meatal eczema, chronic purulent otitis media, chronic accessory sinus suppuration—are positive contraindications to diving and to the submersion styles of swimming. Even though his infection may not endanger others more resistant, an enlightened self interest should prevent the individual from exposing himself to situations from which serious complications may develop, due to invasion from his own particular germs.¹⁹

Individual susceptibility to the action of water upon the skin and mucosal surfaces varies enormously. Manifested by ready chapping, eczema, dermatoses, trichophytal invasion of the toes or fingers, it extends with equal readiness to the epidermal and mucosal linings of the nose and ear in such individuals. Allergic sensitiveness does not apparently predispose to nasal irritability from swimming, unless water enters and cannot readily be expelled during a period of allergic swelling. Under these circumstances water may assist in creating the disagreeable secondary bacterial involvement of vasomotor rhinitis.

In 1922, we were rash enough to offer the suggestion that accessory sinus disease from swimming is largely due to the traumatism of water, causing not only loss of the protective film of mucus, but also maceration and edema of the cellular elements, interference with ciliary action of the mucosal cells and the introduction into feebly immunized accessory cavities of germs from the individual's own highly resistant direct airways.^{12 20} A rather considerable and varied flora is harbored in the inferior and lower part of the middle meatus; the sinuses and sphenothmoid recess are relatively sterile. Studies upon media containing human blood and under partial air tension will demonstrate various staphylococci and streptococci not

recognizable by ordinary methods, which may cause violent trouble in the antrum or sphenoid, or the middle ear, while they cause no reaction and are unsuspected in the lower nose and throat.²¹ Marshall Taylor's exhaustive studies of the effects of sea and tank water,¹⁰ and of body chilling,²² brought clinical confirmation of the experiments of Mudd, Grant and Goldman,²³ who had demonstrated in 1921 that ischemia of the nasal mucosa follows exposure of the body to cold. This is the second factor in reduction of resistance to the individual's own germs. Similarly, traumatism of the nasal vestibule or auditory meatus by water, or water and sand or mud, opens subcuticular avenues for skin staphylococci and molds which are everywhere present and otherwise harmless.²⁴

The untimely passing of Harry P. Finck of Boston has terminated seven years of arduous work under Professors Mosher and Verhoeff which bade fair to solve many of the clinical puzzles of mucosal pathology. His posthumous contribution,²⁵ based on arduous study of every possible type of mucosal disorder, should become a classic in our pathologic literature, and supplies the record of tissue changes to explain clinical facts adduced by Taylor and by us.

Perhaps the only reaction which the normal nose will have to the entrance and immediate exit of water during swimming is the "physiologic inflammation," defined by Fink. This is a mild congestion, with slight submucous eosinophilia, based rather upon vasomotor changes than surface trauma or osmotic damage to cell contents. Let plain water remain in contact with the mucosa, however, and certain cells will be injured by abstraction of their saline content, and will eventually exfoliate and leave gaps into which bacterial invaders may enter.¹² Finck describes the progress of cellular edema and round cell infiltration, and the rôle of the vascular system in combating bacterial invasion. His identification of subepithelial eosinophile cells as indicative of vasomotor instability and allergic susceptibility is of great prognostic importance. He is specially insistent that vasomotor change is prerequisite to bacterial invasion. Unless chilling, ischemia and mucosal loss of tone takes place, bacteria cannot readily enter the intercellular spaces: this is evidenced by the relatively late appearance of polymorphonuclear leukocytes.

We have recently examined a series of sections from turbinal mucosa exposed for stated periods to the action of plain cold water, with very low mineral content, as compared to the same tissue kept in Locke's physiologic fluid at the same temperature for the same periods. One turbinate was a thin crowded middle from an old ethmoiditis case; the other, a pale boggy affair from a case of spring hay fever with secondary antral infection. Pieces were soaked for fifteen minutes and for one hour, then fixed in Orth's solution and stained with hematoxylin eosin. In the control specimens, the only evidence of change from the soaking in physiologic solution was a slight loosening of the submucous layer after one hour. After fifteen minutes in plain water, all specimens showed distinct evidence of imbibition changes, slight in the epithelial layer, marked in the deeper mucosal cells, and very marked in the submucosal layer. There was not much further change after one hour, except that in the already edematous allergic case much greater edema was present after one hour's time. Comparing the epithelial layers, there seemed to be a considerable swelling of the cell bodies and somewhat poorer staining. Actual gross loss of substance in the epithelial layer, where present, was probably an artefact.⁴³ This is, of course, only a preliminary report on this type of examination of tissue, and will be checked further. It is interesting to note that the changes noted agree with our hypothesis regarding cellular death from the toxic action of water, in 1922;¹² also that the subepithelial area, where eosinophiles are found,²⁵ is that showing the greatest amount of edema.

Further changes, such as ethmoiditis and the involvement of the other sinuses, are essentially similar to those of sinus disease otherwise originated; the same is true of the later stages of purulent invasion of the middle ear and mastoid after swimming. Numerous reports on both types of cases stress the fulminating character of the invasion, especially as to frontal bone destruction,^{26 27 28} meningitis,²⁹ orbital abscess, lateral sinus thrombosis,³⁰ and brain abscess in the frontal^{27 31} or temporosphenoidal lobe.³² This may be explainable by the effects of actual hydrostatic pressure, familiar to all divers; as, a squeaking or bubbling sound when air rushes from a sinus at the deepest part of the dive, followed by a sensation

of heaviness or of sharp pain owing to intrusion of the water; also, a sudden rushing or thunder clap as water penetrates to the tympanic cavity through the eustachian tube, followed by almost total deafness until swallowing and autoinflation permit the fluid to run back into the throat. These experiences are common among swimmers, but are not analyzed unless expert questioning is done. It is not improbable that some of the fatalities from so-called "cramps" or from "striking the bottom" may be due to violent labyrinthine concussion and syncope, from the sudden entrance of water into the tympanic cavity. Such a helpless, fainting victim, out of reach at the bottom, is rarely brought out before respiratory collapse has done its fatal work.

There is little wonder that the puny denizen of steam heated apartments, factories and offices becomes subject to ethmoiditis and otitis media when he exposes his dessicated turbinates to the tank water of his Y. M. C. A. or to the breezy tides of an ocean beach. He is a far different specimen from the sleek, well oiled gymnast of imperial Rome, far less resistant than the half clad barefoot boy who trudges across fields to the tempting creek or millpond. It has, therefore, seemed proper to the Committee on Otorhinologic Hygiene of Swimming, appointed from our section of the American Medical Association, to suggest that we direct public attention to the harm which swimmers may do to themselves by entering the water while any obstructive lesion or active infection of the nose or ear is present; also, to suggest that proper methods of breathing and of removing water from the nose and ears be taught to all swimmers. Use of rubber stoppers (or preferably raw or oiled cotton, or lanolin on wool) keeps water out of the meatus, but not the eustachian tube. Duty to our patients includes well considered caution respecting prophylaxis of ailments due to faulty habits in swimming and sea bathing.

Lest it be thought that these opinions are personal, we have recently asked a number of leading men in all parts of the United States for their impressions on these questions. The answers of Doctors Joseph Beck,³³ Chicago; R. C. Lynch,³⁴ New Orleans; E. C. Sewall,³⁵ San Francisco; Hill Hastings,³⁶ Los Angeles; Thos. E. Carmody,³⁷ Denver; A. W. Proetz,³⁸

St. Louis; Ross H. Skillern,³⁹ Philadelphia; Lee M. Hurd,⁴⁰ New York, and Harris P. Mosher,⁴¹ Boston, are certainly authoritative as well as widely separated in climate and geography. Their average number of cases seen yearly, directly attributable to swimming, runs from three to fifty, averaging about twenty. The proportion of children seen varies from 25 to 75 per cent—mainly young adults in Dr. Skillern's series. No distinction as to pathogenesis was made among school tanks, clubs, public pools, park pools, rivers and beach bathing, although some of the worst cases originated from indoor tanks. Of the ear cases most were furunculosis; perhaps 25 per cent had catarrhal otitis; about 35 per cent developed purulent otitis; mastoiditis and other complications were rare. Of the nose and throat cases, nearly all had a simple acute rhinitis, which progressed in perhaps 35 per cent to an acute ethmoiditis. Maxillary sinusitis was rather common, frontal involvement rather rare. Tonsillitis and laryngitis were very unusual. Coincidental eye irritation was noted by about half of the observers, not often needing treatment. Previous nasal obstruction, sinusitis and otitis were thought to be decidedly more likely to have exacerbations after swimming. Dr. Proetz does not feel that hay fever attacks are made worse, while most of the others do. The fact that some persons out of a large group using bathing facilities are alone infected while the rest escape was variously explained. Dr. Lynch mentions dilution of the infecting agent. Drs. Skillern, Hastings and Proetz feel that infection comes from the patient's own germs in most cases. Drs. Hurd and Carmody mention the factor of better individual immunity, coupled with better management of water entering the nose and care to avoid chilling. Dr. Mosher thinks that most purulent otitis cases will be found to have had previous ear pathology. Drs. Beck and Sewall do not attempt an explanation. There is general agreement that crawl swimming and diving are more likely to cause infection than the old breast stroke, and also that the experienced swimmer is less likely to have these troubles than the beginner. Dr. Hurd sees little difference owing to experience, and Dr. Skillern states that some of his worse cases have been in strong and healthy athletes. The factor of carelessness and "showing off" may account for this. Dr. Beck suggests weak phenol-iodin-alcohol

drops as a prophylactic against external ear damage, and forbids manipulation of the external ear after bathing. Dr. Carmody cites a meningitis case wherein infection followed the olfactory nerve fibrils through the cribriform plate. He has seen numerous cases with granular and atrophic pharyngitis. Dr. Hastings affirms his belief that patients rarely get infected from pools but carry their infection with them into the pools..

These distinguished teachers and clinicians are careful to say that their statements are matters of opinion and are in no sense statistical; but they demonstrate rather conclusively the incidence of numerous cases wherein the pathology is traceable to swimming, in all parts of the United States, without much difference based on climate. Attention to the hygiene of swimming, especially as to proper breathing habits and avoidance of hard blowing of the nose, is as necessary along the gulf or in California as in Maine or Oregon or Illinois. Insistence on clean tanks is good, but people should be made to realize not only the danger to others from going in without a good preliminary scrub, but especially the danger to themselves from taking septic noses or ears into clean water. Danger threatens them from their own bacteria driven into unprotected regions; danger from body chilling; danger from water left to macerate delicate surfaces. Perhaps we have blamed germs too much and faulty hygiene too little, meriting somewhat the reproach which Oliver Wendell Holmes, in 1860, addressed to the Massachusetts Medical Society:⁴²

"Disease, being always an effect, is always in exact proportion to the sum of its causes. . . . The one prevalent failing of the medical art is to neglect the causes and quarrel with the effect."

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XXIII.

THE AUDIOMETER AS A DEVICE FOR DETERMINING QUANTITATIVELY THE HEARING FUNCTION IN EACH EAR SEPARATELY.

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The conception that in the physiology of hearing the perception for the several tones in the scale takes place in different parts of the cochlea and the fact that in some diseases of the ear the defect in hearing may be restricted to but a part of the tone scale or when there is involvement of the entire tone scale the defect may be more marked in some parts than in others has made it desirable, in the first place, to be able to test separately different parts of the tone scale, and in the second place, to be able to measure the intensity of the stimulation requisite for producing the sensation of hearing in the different parts of the scale. With this, of course, is the importance of being able to test each ear separately.

Bezold was the first to devise means which made it possible to test separately the hearing for each tone in the entire range of hearing and, in a crude manner, to estimate quantitatively the hearing defects, or rather the residue of hearing for each part of the tone scale. Bezold, together with the assistance of Edelmann, devised as a means for testing the entire tone range a series of tuning forks producing pure tones for the lower part of the tone range, and a series of whistles for the upper tones. By this means Bezold was able to test by air conduction the hearing for each part of the tone scale throughout the entire range of hearing from C_2 (16 d. v.), the lowest tone that can be perceived as a tone, up through the higher ranges of the Galton whistle.

There are two outstanding defects in the method devised by Bezold for testing the hearing. The first is the inability to measure at all accurately the intensity of stimulation required for the perception in the several parts of the tone ranges. The

second is the ever present difficulty of testing each ear separately. The failure in Bezold's method of providing a measure of the intensity of stimulation required for sound perception and the physical limitations of his forks in being able to produce only a modicum of stimulation for a large part of the tone range led Bezold to the false conclusion that actual gaps or defects occurred in the midst of the tone scale.

What actually occurs from diseases of the ear affecting the hearing is a depression of the hearing function, sometimes throughout the tone range but very often involving particularly one end or the other only, or a more or less marked depression in the midst of the tone range, but never does there develop actual gaps or complete defects in the midst of the tone scale, such as one might anticipate were the Helmholtz theory of hearing entirely correct, namely, that isolated areas in the organ of Corti are concerned with the perception of the different tones in the scale.

Bezold worked with the problem of testing each ear separately by endeavoring to determine just what part of his series of forks and whistles could be excluded from the opposite normal ear when testing the ear in which he believed there was no vestige of hearing, the labyrinth having been extruded in a sequestrum. Bezold suggested that the findings in a case of this sort could be used for comparison with the findings obtained in examining patients where total loss of hearing in the one ear was suspected, and in this way it would be possible for one to make a diagnosis of total, monaural deafness.

As a matter of fact, cumbersome as it is, something similar to this is, after all, our surest method of determining the existence of total monaural deafness. The use of the noise apparatus of Bárány is a delusion. In the first place, in order to determine the existence of a substantial remnant of hearing in one ear it is not necessary to use a noise apparatus to exclude the opposite ear, and in the second place, in just those cases where because of the great reduction in the hearing in one ear the question arises as to whether this reduction amounts to total deafness, the noise apparatus of Bárány is not applicable, because by starting the noise apparatus in the normal ear any modicum of hearing that may still persist in the affected side is also drowned out.

It has been assumed by some that the audiometer offers a satisfactory means of testing each ear separately, and estimates have been made in the physics of the earpiece to show that all the hearing that is perceivable when the earpiece is held in place is by air conduction and is, therefore, readily excluded from the opposite ear. The fact that in persons with increased bone conduction the telephone, with the receiver placed against the side of the head, is heard with remarkable clearness, whereas, in persons suffering from deafness, where there is a diminished bone conduction, exactly the reverse is observed, is sufficient evidence that bone conduction actually plays an important part in hearing over the phone. We should expect, therefore, to find that in testing with an electrical audiometer where a similar earpiece to that of the telephone is used for conducting the sound waves into the ear, bone conduction plays a not unimportant part.

In order to demonstrate that bone conduction does take a part in the hearing when testing with the audiometer, we selected a series of cases where we were quite satisfied that the hearing in one ear was totally destroyed, and we found in every instance that there was a considerable degree of hearing still perceptible when testing this defunct ear. That this hearing was conveyed to the opposite normal ear through bone conduction was evidenced, in the first place, by the fact that as soon as the earpiece was lifted from its contact with the head, that instant all hearing ceased. The tones were not being conveyed by air conduction to the opposite side. Moreover, in most of these cases which we tested—over a dozen—the patients lateralized the hearing distinctly in the opposite or normal ear. I have selected only a few of those cases, which I shall report.¹

CASE REPORTS.³

Case 1.—Mr. John M., aged 28, seen by me in April, 1927.² This patient came because of a discharge in the left ear which

1. In order to eliminate as far as possible the element of bone conduction we used a specially made rubber pad on the earpiece.

2. This case is reported more fully in "What Can Be Heard Without a Cochlea and the Problem of Determining Monaural Deafness," *Archives of Otolaryngology*, March, 1925, Vol. 1, pp. 272-276.

3. We used in making the audiograms for these cases a 1-A Western Electric Audiometer.

had dated from an injury received in the coal mines a year earlier and for the relief of which he had already had two mastoid operations. There was a paralysis of the facial nerve on that side. At the time of the accident he not only lost the hearing in that ear but developed the symptom of vertigo, which persisted for some weeks.

In operating upon this patient we found a sequestrum which included a large part of the labyrinth and in which the whole of the basal coil of the cochlea could be identified.

The turning responses were as follows: Nystagmus after rotation to the right very weak, lasting scarcely ten seconds; after rotation to the left, distinctly more vigorous and lasted fully eighteen seconds. We had here a condition which had destroyed the entire labyrinth, the acoustic as well as the vestibular mechanism. The hearing in the opposite ear was practically normal for ordinary tests.

The audiogram is shown in Chart I. Note that in the left ear, which was the affected ear, there was a very considerable margin of hearing preserved throughout most of the tone scale; that there was hearing for the lowest tones, and only the upper tone ranges were dropped out entirely. The patient distinctly lateralized the hearing in the opposite or normal ear.

Case 2.—Mr. Frank P., aged 39, consulted me in December, 1921, because of a chronic running ear of long standing. For some weeks previous he had had more or less disturbance from slight attacks of vertigo, and we had no difficulty in demonstrating more or less typical fistulous symptoms by gentle compression of the air in the external meatus. The condition was one of cholesteatoma. Several weeks later he came a second time to the dispensary complaining of an intense headache and annoying tinnitus in the affected ear. The temperature was 102 F.

The Weber was lateralized to the opposite or normal ear, fistula reactions were gone, the patient had a marked increased cellular count in the spinal fluid, and a diagnosis of a suppurative labyrinthitis with threatened meningitis was made. A radical mastoid with exenteration of the labyrinth was at once carried out. The patient recovered and has been under observation off and on ever since.

The nystagmus after rotation to the right is very weak and lasts five seconds, and after rotation to the left twelve seconds, much stronger. Weber is lateralized to the normal ear, and there is total deafness in the side on which we carried out the exenteration of the labyrinth.

The audiogram of his hearing is shown in Chart II. Note the practically normal hearing in the right ear and the hearing curve for the left ear, which follows more or less the curve showing the hearing in the opposite ear.

Here are two patients where not only could we diagnose complete destruction of the labyrinth from our functional tests, but we had surgical demonstrations that the labyrinth had been destroyed, yet the patients hear distinctly with the audiometer throughout a large part of the scale.

Case 3.—Mr. Joseph B., aged 52, consulted me in September, 1927, because of defective hearing and tinnitus on the left side, which resulted from a fall from a wagon four weeks previous, the patient striking his head on the pavement, was unconscious from the accident and bleeding had developed from the left ear. The functional tests gave no evidence of any remnant of hearing in the affected ear by the ordinary tests. The nystagmus after rotation to the right was four seconds and very weak. After rotation to the left, nystagmus lasted ten seconds and was much stronger. Caloric responses from the right ear were prompt; after syringing the left ear with ice water for four minutes there was no response.

Here was a patient where only one conclusion was possible, namely, that he had sustained in falling from his wagon a fracture through the base of the skull, passing through the petrous bone, and, like such fractures, the injury had destroyed totally and permanently the entire function of the internal ear.

The audiogram of his hearing is shown in Chart III. Note that the hearing in the right ear is practically normal and in the left ear there is what would ordinarily be designated as a considerable remnant of hearing throughout the entire tone scale, except for the very highest tones, yet the patient lateralized the hearing distinctly in the opposite ear. We had clearly a case of monaural total deafness.

Case 4.—The fourth case is a boy, Paul G., 12 years old, whom I saw in November, 1926, when he consulted me because of deafness and tinnitus in the left ear, which dated from an injury sustained in August, 1925, when he was struck by a truck. The history discloses the fact that immediately following the accident there was bleeding from his ears as well as from his nose, he was extremely dizzy and was confined to his bed on this account for two weeks, and since the injury he has suffered from deafness in the left ear and a buzzing tinnitus. The responses from the functional tests for hearing as well as for the vestibular mechanism were typical for those obtained where the labyrinth has been destroyed.

The audiogram of his hearing is shown on Chart IV. The hearing in the right ear is normal, and there is not much more than a 50 per cent loss of hearing in the left ear which, however, the patient lateralized distinctly in the right or normal ear.

Case 5.—Mr. A. H. consulted me in February, 1918, because of a persistent discharge from the right ear and for which he had had a mastoid operation two years previous. Our examination found no vestige of hearing for the ordinary tests. This patient had defective hearing in both ears. The Weber was lateralized to the left, which was his better hearing ear. As far as our functional tests could determine, there was no vestige of hearing in the right ear. The turning tests gave a weakened nystagmus, only thirteen seconds' duration, after rotation to the right, and the same type of nystagmus, eleven seconds, after rotation to the left. It was not feasible to carry out the caloric reactions, because of the discharging ear, but all of our functional tests bore out the conclusion that there was a dead labyrinth on the right side.

Chart V shows the audiogram of his hearing. In the left ear there is a defect in hearing throughout the tone scale which increased somewhat for the highest notes but is greatest in the midst of the higher tone range. In the right ear the audiometer was heard throughout a large part of the scale but always lateralized to the opposite ear. Note that the curve for the left ear follows more or less closely the general outlines of the curve for the opposite ear.

CONCLUSION.

The conclusion seems clear that the audiometer cannot be used as a means for determining the hearing in each ear separately, especially when there is a marked defect in the hearing, for the obvious reasons that where it is necessary to increase the intensity of stimulation in order that the patient may hear, bone conduction becomes a factor, and where bone conduction is a factor in hearing it is not possible to examine the two ears separately.

In looking over a large series of audiograms made from patients where there was an extensive defect involving both ears we found that the audiograms for the two ears invariably tend to follow parallel curves, a fact which demonstrates that bone conduction is an important factor and that the two audiograms are largely a record of the hearing in the better hearing ear.

The audiometer supplies a means of testing quantitatively the hearing in the several parts of the tone range, which is an improvement over previous methods. The facts brought out by this method of testing the hearing are of interest, however, especially to those who are interested in the problems concerning the physiology of the hearing. There seems to be very little gained by an examination of the audiograms that is of practical use to the otologist—that is, the ascertaining of those facts which have a practical significance.

For example, a defect especially for the low tones is recognized as having an important clinical bearing for the practicing otologist. The presence of such a defect is more readily and more accurately determined by tuning forks, for with these it is possible to test this part of the tone scale in each ear separately by air conduction, whereas, in using the audiometer bone conduction is not excluded, and just in these cases where there is a defect in the lower tone ranges only the audiogram gives misleading conclusions because of the heightened bone conduction.

Again, a defect, especially at the upper tone limit, has another quite important clinical significance. This defect, too, is readily ascertained by means other than the audiometer.

There is one fact brought out in the audiogram that cannot be easily obtained by other methods. This is the quantitative estimation of hearing defects in the midst of the tone scale, but ascertaining this fact is of relatively slight clinical significance compared with defects at the upper and lower tone limits.

We have carried out a large series of tests with the audiometer in order to determine what practical value it may have in determining fluctuations in hearing, and we shall give a detailed account of these tests in a later communication.

There has been in some quarters an attempt to utilize the audiogram for estimating the percentage loss of hearing in forensic cases. This is unfortunate, since the percentages of loss in hearing, as shown in an audiogram, bear no relation to the actual handicap that may result from a partial loss of hearing. It is the voice tests alone that have any practical value. For example, a person may show a percentage defect in an audiogram amounting to, say, 70 or 80 per cent, but if this defect be chiefly in the tone range outside that part which is used for hearing the spoken voice there may be an actual handicap, represented more accurately by 5 or 10 per cent. The audiometer is a mechanism of some value for studying the physiology of hearing, but so far has been shown to be of very little practical value to an otologist.

CHART I.

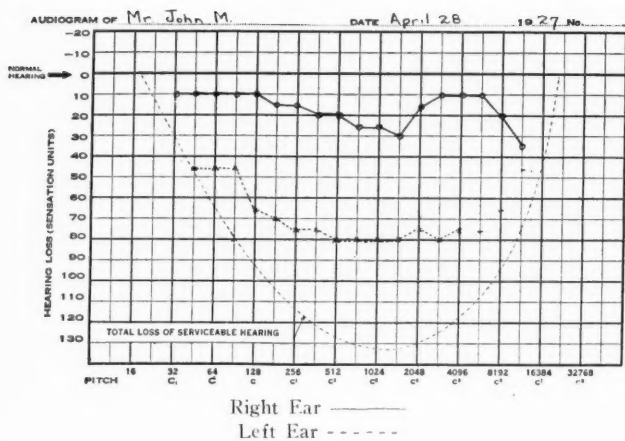


CHART II.

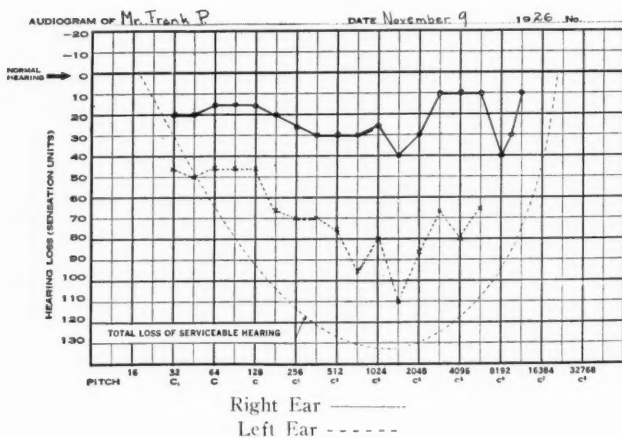


CHART III.

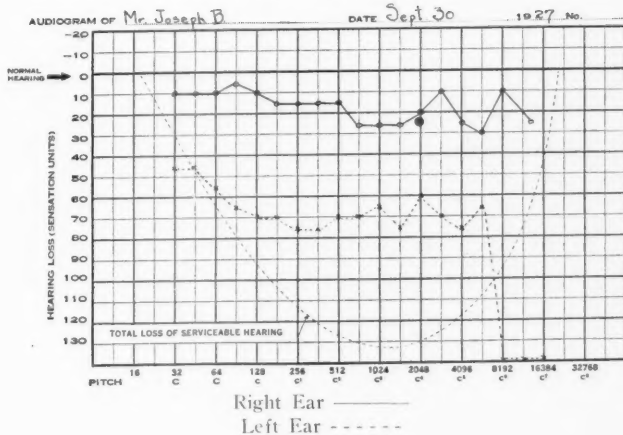


CHART IV.

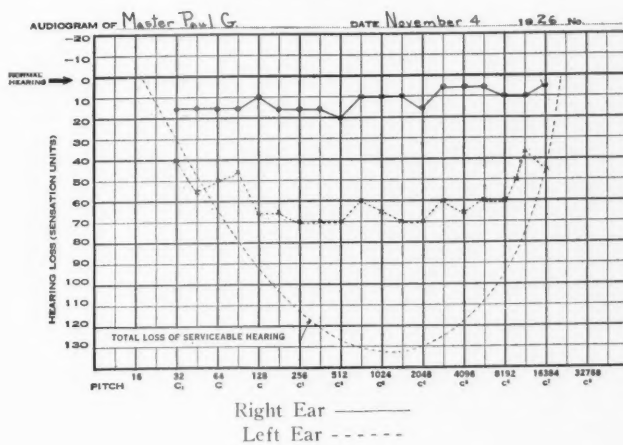
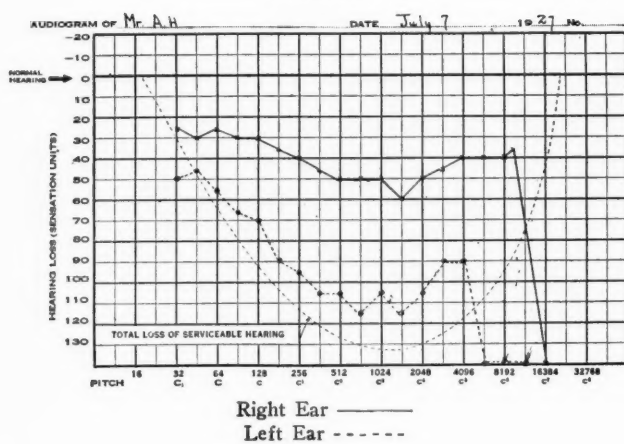


CHART V.



XXIV.

OTOSCLEROSIS, NONSPECIFIC PROGRESSIVE DEAFNESS: OTITIS INSIDIOSA.

By W. SOHIER BRYANT, A. M., M. D.,

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Otosclerosis, nonspecific nerve deafness and all other forms of progressive deafness, except nerve deafness of specific origin, are amenable to treatment. This conclusion and the reasons for it are founded on extended clinical experience with these diseases.

Under the comprehensive term of "otitis insidiosa" the author groups all forms of nonspecific, progressive deafness.

Diagnosis: The functional symptoms in otitis insidiosa—nonspecific progressive deafness—include loss of the two ends of the tone scale, paracusis of Willis and any other auditory defect, either quantitative or qualitative. In advanced cases the diagnosis is easily made by the suggestive history of failing hearing extending over a considerable period and with the characteristic functional tests of middle ear, labyrinthine and nerve deafness, but without suppurative otitis, past or present, and without evidence of any specific etiologic factor.

The characteristic flushing of the promontory that is sometimes seen in otosclerosis, and especially in the early stages, has corroborative diagnostic value. Another symptom that is valuable in making the diagnosis a symptom which is often present in the neuritic variety of otitis insidiosa, is the more or less marked anesthesia of the fifth nerve, more pronounced on the side of the worse ear symptoms. (Froeschels.)

Also otitis insidiosa may be present, even when there are other ear diseases, and, naturally, these complicating ear diseases confuse the diagnosis.

An early or differential diagnosis is based chiefly on the question of heredity, but especially on the instability of the endocrine system. It is very difficult to distinguish the early cases of otitis insidiosa from the cases of deafness which will

run their course without developing any of the forms of otitis insidiosa. The diagnosis often has to be made by exclusion of the acute conditions of the ear causing impaired hearing, and further by elimination of the effects of middle ear suppuration and specific nerve deafness. Often a considerable time for observation of the case is required before a definite diagnosis can be made.

Pathology: The pathologic changes in the progressive, non-specific deafnesses—otitis insidiosa—are all grouped under one heading: Degeneration. Each tissue is individual and invariable in its pathologic reaction, but there is variation due to the particular tissue affected at a particular period in the progress of the disease. The pathologic changes are nonspecific in character and may arise from a variety of causes. (Fraser.)

Predegenerative circulatory pathology, which later leads to degeneration, must be kept in mind, because at the onset of the disease these changes are the precursors of the later degenerative changes found in the cases of long standing.

In otitis insidiosa, all parts of the hearing mechanism are subject to attack. The pathology includes all variations that are found in chronic middle ear deafness that is not specific or suppurative in origin.

Etiology: The etiology rests upon three points, namely, focal infection, endocrine dysfunction and hereditary tendency.

The focus of infection is usually located in the region of the pharynx, where the most important point is the pharyngeal bursa. Clinical experience proves that an activating factor in the form of a focal infection can act only on a ground previously weakened by defect of the endocrine system.

Endocrine deficiency is commonly in evidence, and various glands have been selected by various authors as especially blameworthy. (Warwick and Stevenson, Drury.)

The pituitary is, in my opinion, the chief defective link in the glandular system. (Denker.) This conclusion is supported and justified for the following reasons: First, the pharyngeal pituitary, homologous with the cerebral pituitary in origin, is situated close to the pharyngeal bursa, which is the usual seat of the activating focal infection of otitis insidiosa. (Citelli, Poppi.) Second, the bony changes and other tissue changes of otitis insidiosa are identical with tissue changes known to

be caused by a diseased pituitary. (Nicolai, Pisano.) Third, the most common cause of exacerbations of otitis insidiosa is generally acknowledged to be pregnancy, during which time the cerebral pituitary is often abnormal. (Blohmke.) Fourth, the heredity factor in the etiology of otitis insidiosa is a matter of general observation, and this hereditary tendency is further emphasized by the recognized heredity of endocrine derangement. Fifth, much of the tinnitus accompanying otitis insidiosa is muscular in origin and is due to spasm of the intratympanic muscles. These abnormal muscular contractions are doubtless caused in part by derangement of the pituitary in conjunction with the thyroid and parathyroid glands, since it has been observed that tetanic muscular movements follow the derangements of these glands. (Frey and Orzechowski.)

Prophylaxis: Prophylaxis demands that special attention should be paid to those individuals who have hereditary predisposition to progressive deafness, or who have a weak endocrine system or focal infection. We should at once remove the possible cause in predisposed individuals, especially in the patients who show a bad family history. The road open to the elimination of progressive deafness—otitis insidiosa—is not through the impossible dream of rejuvenation of lifeless, degenerated tissues, but along the sure and definitely indicated road of prophylaxis.

Treatment: When we recognize, in cases of otitis insidiosa, the etiologic factors and their sequence and interactivity, the aim and direction of the treatment are clearly and definitely indicated.

All details of the treatment in otitis insidiosa should be directed toward:

1. Elimination of the activating focus of infection.
2. Improvement of the endocrine function.
3. General constitutional treatment, as thorough as possible, and continued for an adequate period of time.

Major and minor operative procedures are required to remove all the foci of infection that can be removed, and local applications are needed for the treatment of the foci that cannot be removed surgically.

Unsatisfactory results of treatment, as a rule, can be laid to the failure to remove the foci of infection. When the focus

cannot be eliminated, it is necessary to continue treatment in order to hold in check the ear degenerative processes.

Prognosis: In cases of otitis insidiosa where no treatment is given, the hearing tends to decrease gradually between irregular remissions of more or less markedly rapid loss, until all useful hearing is completely lost. But approached from a sound and reasonable etiologic point of view, an etiologic point of view that is thoroughly justified, otitis insidiosa need no longer have the hopeless prognosis which has been associated with it. Indeed, the prognosis in otitis insidiosa has, because of the more definite etiology and more intelligent direction and adaptation of treatment, become quite encouraging. The results of the treatment include arrest of the progress of deafness or least a slowing down to an almost imperceptible deterioration, with complete cessation of remissions or exacerbations of the failing hearing. There is also an immediate improvement in the hearing level up to about the best reached during recent periods of intermission in the poor hearing. An important part of the prognosis depends on the ability to eliminate the activating focal infection, as well as to control the underlying endocrine deficiency.

A less optimistic part of the prognosis is the difficulty in eradicating the inherited and congenital defects. Also, the inability to completely restore the hearing function. The damaged degenerated tissues cannot, of course, be replaced or restored. Prognosis depends also on the ability to guide and control the patient and keep him under observation long enough to follow up the treatment till all remissions of the poor hearing have ceased for at least five years. Our point of view does not in any way guarantee that the intercurrent major physical accidents of life may not cause a recurrence of the process of otitis insidiosa in cases where it had completely ceased. This recurrence may be expected as the result of an endocrine catastrophe or a breakdown of normal resistance to bacterial infection. Since the fundamental activating cause cannot always be permanently eliminated, its baneful activity may be renewed under the influence of some great physical strain, such as child bearing, a general infection or the endocrine stress of emotional crises.

Should the process recommence, a speedy control may be assured through knowledge of the patient's constitution already acquired, assuring an early arrest of any breakdown. In spite of the elimination of the otitis insidiosa, it must be borne in mind that the original soil which was capable of harboring such a process may still remain susceptible.

Nomenclature.—Otitis insidiosa is the general, comprehensive term under which should be grouped all forms and varieties of nonspecific, progressive deafness. The term "otitis insidiosa" is of apparent value and significance because of its appropriate literal descriptive meaning. It also has a historical standing, for it was the term used at one time to indicate a kind of case that was later called by the misnomer "otosclerosis." (Ballenger, W. L., and Ballenger, H. Ch., in "Diseases of the Nose, Throat and Ear, fifth edition, 1925, p. 746.)

Since all forms of nonspecific progressive deafness have the same etiologic foundation, and since they are amenable to the same treatment, it is logical and sensible to regard them as different manifestations of the same degenerative process—otitis insidiosa.

If, besides this general, comprehensive term of otitis insidiosa, it is advantageous to use a more specific term also, we may describe one of the included varieties and indicate the seat of the process in the individual case; for example, otitis insidiosa tympanica, otitis insidiosa neurotica, otitis insidiosa labyrinthia, otitis insidiosa cochleæ, otitis insidiosa stapedis, otitis insidiosa fenestralis, and so forth.

If the term otitis insidiosa is used to include all forms of nonspecific progressive deafness, a great deal of confusion concerning the nomenclature of the various subdivisions will be removed. Formerly—and too often now—the classification of the progressive deafnesses has been based on difference, on diversity of the tissues involved and on the different degrees of pathologic change.

A classification founded on similarity is more reasonable and serviceable. In these cases of nonspecific progressive deafness there is an identical etiology, a parallel course, and, in all, the presence of degeneration as the major pathologic factor.

The following may be taken as an illustrative case of the course and prognosis in otitis insidiosa under treatment. A woman, 27 years old, with rapidly failing hearing and distressing tinnitus.

History: Slight thyroid insufficiency, occasional muscular pains, sore throat and symptoms of intestinal intoxication.

Examination: Slightly hypertrophic pharyngeal lymphoid tissues, infected faucial tonsils, abscessed teeth, normal drum membranes, rosy blush on both promontories, patent eustachian tubes, negative Rinne and negative Gellé, and retraction of both ends of the tonal scale. Hearing acuity, watch, both ears 1/100 to 1/200.

Treatment: Tonsils removed and bad teeth extracted. Thyroid gland stimulated and intestines regulated. Special attention paid to the pharyngeal lymphoid tissues remaining after operation. Repeated applications of various antiseptics to the nasopharynx.

Results of treatment: The acute symptoms were immediately relieved, and further deterioration of hearing was arrested. An important degree of improvement of the hearing was obtained, and the general health was markedly benefited. The promontorial blush permanently disappeared after three years of treatment. At the commencement of treatment there were temporary remissions of the patient's deafness, and these remissions gradually decreased in severity and frequency, while the intermissions increased until the remissions ceased altogether. The tinnitus was wholly relieved. Treatments were given at first twice a week, later once a week, then twice a month, and finally only when the patient fancied she needed treatment. During the last five years the patient has had eighteen treatments only. She has been under treatment and observation for eighteen years.

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THE RATIONALE OF TUNING FORK TESTS.

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A definite diagnosis is needed before prognosis can be decided or treatment instituted, and to arrive at the diagnosis careful examination is essential. To do this properly, definite methods are necessary, and one must decide on a regular routine to get comparable results. Instruments of precision are required and they must be properly handled in order to obtain accurate results. The psychology of the patient is important, as the hearing tests are subjective and not objective. It is, therefore, important to note whether the individual is cooperating, whether he is overanxious and tries to anticipate findings, or whether he is simulating disease and thus thwarting the test in every possible way.

In other papers I have described in considerable detail the various methods employed in the testing of hearing. These will, therefore, be mentioned very briefly, more space being given to the discussion of tuning forks, their physical properties, their method of employment, and the interpretation of the results thus obtained in functional testing. A complete otoscopic examination, including that of the auricle, external auditory meatus, drum membrane, as well as inspection of the nose, nasopharynx and pharynx is necessary. A pathologic change in any of these structures may have considerable bearing upon the hearing, or rather the impairment thereof, particularly where some type of conduction apparatus involvement is present. Toxemia arising from disease, such as dental apical abscesses, infected tonsils, suppurating nasal accessory sinuses or other foci in the body may have an injurious effect upon the perceptive apparatus, namely, the inner ear and auditory nerve. Care should be taken in noting the general appearance and conduct of the patient, such as his close observation of the speaker (reading his lips), inclination of the better ear to-

wards the speaker, and the loudness of the patient's voice. When a conduction apparatus involvement is present, autophonia is often present, so that his voice sounds very loud to himself, and in order not to offend, he speaks very low. On the other hand, where there is a marked perception apparatus impairment, the bone conduction is diminished, the individual has the impression that he is speaking very low, and in order to make himself heard he is inclined to speak very loud.

Hearing is usually tested by means of tones, noises, or a combination of the two. A tone is sound produced by a number of equal and regular vibrations, while noises are sounds due to irregular vibrations, irregular both in length and in time. The use of the Politzer acoumeter or the ordinary watch carries with it a number of fallacies. In the first place, the acoumeter is rather high pitched, and with it only one sound can be produced, so that if the individual is deaf for the low tones, this fact cannot be elicited by this instrument alone; and, secondly, the acoumeter produces a noise, not a tone. The watch is not an accurate instrument, since no two watches have the same pitch or intensity; and second, noises and not pure tones are produced. The human voice contains both tones and noises. Though this fallacy is present, as well as the fact that neither the intensity or the pitch can be accurately regulated, nevertheless, testing with the voice is most important, for after all, the criterion of the practical efficiency of the ear is its ability to understand the human voice. Bezold originally assumed the speech area to be very limited in extent, but we now know that the voice sounds range from about 200 to 3,000 double vibrations, the highest overtone of the vowel *e* being at the latter pitch. If an individual can hear all low tones below 200 and all high ones above 3,000, but cannot appreciate those lying within this range, he is unable to understand the spoken voice, and is thus deaf, for all practical purposes, so far as human intercourse is concerned.

In the English language, the numerals are low pitched except six and seven. Unaccentuated whisper or conversation should be employed; the patient should stand at the farther end of the room with the eyes closed or averted, the listening ear facing the examiner, and the other ear closed with the

moistened finger. No part of the body should be allowed to touch the wall or furniture, as the sound may then be transmitted by bone conduction to the skull. When using the voice, the acoumeter, watch or tuning forks, or any other instrument, the source of sound should be brought from afar to the point at which the patient first begins to hear or understand, and this distance noted. If the sound is produced near the ear and then removed farther and farther, the memory of the sound will cause the patient to think he hears further than he actually does. Low tones and high tones are used separately, and the distances at which they are heard noted, or a combination of low and high tones may also be employed. In an open unconfined space, the intensity of sound varies inversely as the square of the distance. In a room or other enclosure, this rule does not accurately apply, since there is a reflection of sound from the walls, furniture and other fixed objects. In other words, if an individual hears one-half the distance that is supposed to be normal, that does not mean that his hearing is reduced one-half, as it may be reduced to one-quarter or some other fraction. However, a good relative idea may be obtained of the state of the hearing by noting the distances at which the various sounds are heard.

Tuning forks are the instruments perhaps most often used for testing hearing. From the prongs we get transverse vibrations of great amplitude but very slight intensity; while from the stem of the fork arise longitudinal vibrations of great intensity but very slight amplitude. They are usually constructed of steel, either nickeled or unnickeled. The disadvantage of nickeling is that when the latter peels, adventitious sounds are produced by the vibration of the loose pieces of metal. On the other hand, the nickeling prevents rusting; for unless unnickeled forks are carefully handled, and dried after using, or are occasionally covered with some oil, they will rust and the pitch will be changed. The Committee on Standardization of Tuning Forks and Tuning Fork Tests of the American Academy of Ophthalmology and Otolaryngology, on which the writer has had the honor of serving for some time in association with Drs. L. W. Dean, George W. Mackenzie and E. G. Gill, have endeavored to find a metal which would be rustless or rustproof and at the same time have the elastic qualities like

that of the steel used in tuning forks. Quite recently a fork made of an alloy in which magnesium constitutes about 99 per cent, has been made at the Riverbank Laboratory, and it is quite probable that this material will solve the problem. These forks are very light in weight, as compared with steel, which has a specific gravity about 7, that of aluminum 2.7, while that of magnesium is about 1.7. The cost of manufacture is about the same as that of the steel forks, and the vibration period of these forks also resembles those commonly used. However, thus far at least, magnesium forks cannot be produced at the very lowest pitches, and another disadvantage is the fact that in testing bone conduction they do not seem to be quite as serviceable as those made of steel. The cause for the latter fact is that the magnesium is not heavy enough to produce sufficient vibration of the cranial bones. Action and reaction are equal and opposite in direction, according to the laws of Newton, and the magnesium alloy is not able to give sufficient reaction of the skull mass, so most of the energy goes back into the fork. Other experimentation will be necessary to determine whether these magnesium forks have all the desirable and essential qualities so that they will be able to displace steel forks. When certain sounding bodies, such as steel wires, etc., are struck, they give forth not only the fundamental tone but several overtones or harmonies are produced. In most instances, the first overtone (which is the loudest) is an octave above the fundamental, and the next harmonic is the fifth above that, and the third is the second octave above the fundamental. Besides these, there are also, as a rule, two or three other overtones. With tuning forks, however, this rule does not strictly apply in that the first overtone is often several octaves above the fundamental. When the first overtone is very loud and shrill, it tends to drown out the fundamental. In carefully made forks, the first overtone lasts but a few seconds, and the fundamental is then heard clearly throughout. It is, therefore, necessary to buy first-class forks if one wishes to do proper testing.

Aside from being properly constructed so that the first overtone is not very loud or long standing, and that rustproof metal be used, if possible, among other essentials which the committee believes good forks should possess are these: (1) They

should be made of one piece of metal; (2) the handle should be long enough so that it can be loosely held without damping or stopping the vibrations; (3) the fork should sound for a long time; (4) and there should be weights on the lowest pitched forks in order to remove overtones. Furthermore, by having the fork properly marked, the weights may be moved along the prongs so that a range of five tones can be elicited from one particular fork. This is a great advantage, in that one does not need to possess so many forks in order to produce the various tones of the lower and middle ranges. Well made forks are the only ones which give satisfactory findings, and I believe it is better to possess four or five first-class forks with which the tests may be properly performed than to have a great number of poorly constructed forks with a short duration of vibration, accompanied by shrill, long lasting overtones. Forks are usually made in a range from C-2 (16 double vibrations) to c-5 (4,096 double vibrations). Forks as low as 12 double vibrations and as high as 14,000 or 16,000 double vibrations have been produced, but are not feasible for ordinary work, are rather difficult to obtain and expensive. Few individuals are able to hear below 16 double vibrations, especially in noisy cities, and for high tones above 4,000 or 5,000 double vibrations. Whistles or the monochord may be employed for testing high tones.

The Committee of the American Academy of Ophthalmology and Otolaryngology believe that for doing the ordinary or usual tests it is not necessary to possess more than five or six forks, and for this purpose recommend the C-1 (32 double vibrations), C (64 d. v.), c-1 (256 d. v.), c-2 (512 d. v.), and c-5 (4,096 d. v.). For the Weber and Schwabach tests many individuals prefer the Bezold large unweighted A (108 d. v.) fork, while for the Rinne test as well as the Stenger test Bezold's unweighted a-1 (435 d. v.) fork is preferred.

For exciting the forks it is best to decide upon one definite routine, so that the results obtained may be fairly uniform and comparable to those made in other cases. The general rule is that the lower the pitch of the fork, the softer should be the material used in striking or exciting it. For the very lowest ones, the side of the hand (hypothenar eminence) may be

used: for the medium pitched, a rubber tipped pleximeter, and for the highest forks a metal hammer. A pendulum of metal covered at the end with rubber gives accurate and uniform results, since it can be made to fall each time through the same arc, thus giving the same intensity of impact. At about the junction of the distal with the middle third of the prong is the so-called "percussion point," and it is advisable to strike the fork in this area, as experiment has shown that when this is done there is the greatest duration of vibration with the least overtones. The larger unweighted lower pitched forks may also be excited by holding them at right angles to the thigh and allowing them to fall through an arc of 90 degrees, striking the flat or outer surface of the prongs against the knee. The higher pitched forks may be excited in three ways, giving varying degrees of intensity, namely, by rubbing the prongs with the finger, striking the prong with the finger nail, and lastly, as previously mentioned, with a metal hammer.

Each fork has its own rate of decrement or decay, and there is only a very little difference in this rate with any one fork unless there is a very marked difference in the force with which it is struck. Forks have the greatest intensity immediately after they are struck; this drops very fast for the first few seconds, then more slowly, in a logarithmic curve, and not by geometric progression. The rate at which the fork dies down is known as the "constant" or decrement or damping. The Bureau of Standards at Washington, D. C., for a fee of \$2 per fork, will determine for each fork the decrement, and by means of this figure it is then possible to determine the percentage of loss of hearing in sensation units, just as is done with an audiometer. In this manner, the same sort of a curve may be obtained. If a good many pitches are tested the process will be considerably slower than with the audiometer, but the results will be just as accurate. The method employed is to determine how long a particular fork is heard by the normal individual, or, if the examiner has normal hearing, he may be used as a criterion. In any case, the number of seconds that the patient hears the fork is deducted from the normal average for that fork, and the remainder multiplied by the "constant" or decrement, which then gives the percentage of loss of hearing figured in sensation units.

During the past few years audiometers of various types have been used considerably, but they have not met with the universal adoption which tuning forks have been accorded. They are instruments of precision and therefore most desirable, but when the tuning forks are properly used they also give accurate results, as previously noted. Audiometers possess many advantages, but have certain disadvantages: first, their great cost; second, the fact that bone conduction cannot as yet be well determined with them; third, that the very lowest and very highest tones cannot be tested with audiometers except with the most expensive types; and fourth, that the curves in many cases do not enable one to make the accurate diagnosis afforded by forks, clinical examination, etc. Audiometers are very useful and especially so in research work, and I believe they should be further studied and used. For all practical purposes, however, by means of the voice, the use of five or six well selected forks properly applied, with whistles and monochords for the high tones, if all tests are well done, a diagnosis may be made with this comparatively inexpensive armamentarium. By means of resonators, especially the simple contact set devised by Prof. Karl L. Schaefer, it is possible to amplify sounds to see whether a residuum of hearing is left, and also to determine the pitch of a tinnitus which is often of great use in differential diagnosis of conduction mechanism and perception apparatus impairment; with the former there is often a low tinnitus, and in the latter condition there is usually a high pitched one. Resonators, of course, cannot amplify sounds at all pitches to the extent that an audiometer does, but nevertheless, with a pitch range between 100 and 1,000 double vibrations, they are often of great use in determining the practical degree of hearing present. Resonators show their great efficiency in the middle octaves, being much less powerful at the lowest and highest pitches.

Quite a number of tests have been devised from time to time, such as the Weber, Schwabach, Rinne, Politzer, Stenger, Gelle, Bing, and the Lucae-Dennert, etc. Of these the most important, I believe, are the Weber, Schwabach and Rinne. In determining the simulation of deafness, the Stenger test is extremely useful, but the other tests mentioned are not necessary, as a rule. The purpose of functional testing is to

determine the presence of an impairment of hearing and the degree thereof, as well as to decide the location of the lesion, whether it is in the conduction apparatus, the perception apparatus or in both. For these purposes, the Weber, Schwabach and Rinne are most essential.

The Weber Test.—This test is used for determining the presence of the lateralization of sound. As a rule, one of the heavier low pitched forks is used for this purpose. The A (108 double vibrations) fork is the one we are in the habit of employing. After striking it, the fork is placed on the median line of the vertex, or, as some do, at the root of the nose, on the teeth or the chin. When applied to one of the latter positions, the tone is usually heard louder, because the mouth and nasopharynx act as resonators. The patient is then asked whether he hears the fork louder in the head itself or in one of the ears.

Interpretation of This Test.—Normally, with both ears in the same condition, the sound is usually appreciated "in the head" of the patient. If there is a conduction apparatus impairment, the sound is usually heard in the worse ear, but with a perception apparatus involvement present the sound is usually heard in the better ear. There are, however, many exceptions to this rule. The degree of lateralization may be noted by shifting the fork away from the median line of the head. If heard in the left ear with the fork held on the median line, it may be shifted somewhat towards the right side of the head to see how far from the median line it may be moved without influencing the lateralization to the left ear. If heard in the left ear, with the fork far over to the right side, there is a very marked conduction impairment in the left ear or a very decided perception change in the right ear. However, in this connection one must remember that if a tuning fork is placed laterally to the median line of the head, diagonal resonance or crossed perception of sound is noted. When placed on the median line, the head is divided into two equal parts, both of which apparently resonate equally well, but when the fork is shifted to one side of the median line the skull is divided into two unequal resonating masses, the smaller of which is the tone on which the stem of the fork is resting and the larger is the contralateral side. For instance, if a fork is placed external

to the median line, on the right side of a normal individual, the sound is heard louder on the left side than on the right, and vice versa.

The Weber test is also of considerable aid in differentiation between an external otitis and otitis media, especially in young children. Where there is considerable difficulty in otoscopic examination the lateralization of the Weber is of considerable aid in differential diagnosis. We usually have a lateralization with an otitis media but not, as a rule, with otitis externa. A very important phenomenon is the change of the lateralization from one side of the head to the other. If, for instance, in the course of a left sided acute otitis media (which is a conduction apparatus impairment), with the Weber heard in that ear, there is suddenly a change in the lateralization of the opposite or right ear, it usually means that there has been an extension of the process, with involvement of the inner ear (labyrinthitis), and then the Weber is heard in the right or well ear. A change of the lateralization is thus very significant of an extension of the pathologic process from the middle ear. With the exception of the change in lateralization, we may say that, as a rule, the Weber test is of real value only when it agrees with and confirms the other clinical findings.

Schwabach Test.—The test is employed for determining the duration of the bone conduction of the individual as compared with the normal standard. While it is often assumed that this test is always done with the fork on the midline of the vertex, as in the Weber test, many otologists actually place the fork on the vertex or the mastoid process or both. We believe that a rather low pitched heavy fork (Bezold's unweighted A, 108 d. v.) is well suited for this test, provided the fork is to be used on the vertex. If the bone conduction factor of the Rinne is used in place of the usual Schwabach, it is obtained by placing the fork on the mastoid process and then a smaller and higher pitched fork, such as the C-1 (256 double vibrations) and the Bezold a-1 (435 double vibrations), seem to serve better. Where the Schwabach test is done in the old classical manner, after excitation of the fork, it is placed on the median line of the vertex, seeing to it that the hairs are separated so that the fork rests directly upon the scalp. The patient should be cautioned against confusing the feeling of the vibrations of the fork with

the hearing of the actual tone thereof. The fork should be allowed to rest, if possible, of its own weight, since the pressure with which it is applied changes the amount of the bone conduction. Lastly, the stem of the fork should be held not too tightly, as the vibrations are thereby damped or stopped. When the patient states that he no longer hears the sound, remove the fork for a second, because of the so-called "fatigue symptom," and then replace it upon the head and note whether it is again heard. When no longer perceived, the fork is set upon the head of the examiner or some other individual whose bone conduction is known to be normal. In order to make this test more objective and not dependent upon some other individual, I have been in the habit of determining the average duration of hearing for the particular fork employed, in a large number of ears. Having obtained this figure, simply compare it with the length of time which the fork is heard by the patient, and thus at once see whether the bone conduction is lengthened, shortened or normal. Bone conduction, even in a normal person, varies considerably, depending upon age (at or after middle age it is considerably diminished), and it may differ in persons of the same age. Other factors influence the duration of bone conduction, such as the thickness of the cranial bones, the size of the air spaces of the mastoid cells, as well as certain anomalies of the skull, such as traumatism, adhesions between the dura and the bone, the amount of the hair, the tension of the skin, the degree of contact and the pressure with which the fork is applied. It is necessary, however, to remember that a slight increase or diminution in bone conduction does not have any definite significance. Politzer states that from a prognostic point of view cases of lengthened bone conduction are usually more favorable in their course as a result of treatment than when it is shortened. We must not forget that a typical otosclerosis, which gives prolonged bone conduction, has a very poor prognosis. On general principles, one may say, bearing in mind the various factors which are mentioned, that lengthened bone conduction usually means impairment of the conduction apparatus, and diminished bone conduction usually indicates some form of inner ear or auditory nerve lesion. Dozent Beck of Vienna called attention to the diminution in bone conduction at times noted in cases of lues with good hearing and

no aural symptoms. He says that this lowered bone conduction is present in about 80 per cent of all syphilitic subjects, and that it appears mostly in the second and only rarely in the first stage. We have found this strikingly illustrated in a number of cases of rather early lues where the hearing was still apparently very good, the bone conduction much diminished, and the Wassermann test found strongly positive. I believe that a careful study of bone conduction is essential for diagnosis in many ear conditions, and a careful performance of this test is therefore of great importance.

Rinne Test.—This is employed for comparing air conduction with bone conduction in the same individual. This test is usually made by setting the vibrating fork on the mastoid bone in the region of the antrum, and when the sound is no longer heard holding the prongs near the external auditory meatus and observing how much longer it is perceived by air. Care must be taken to see that there is no contact with the auricular vibrissæ, otherwise there is damping of the fork. In the normal individual air conduction is very considerably longer than bone conduction—that is to say, the Rinne is “positive.” With various forks used in a long series of cases, I observed that the ratio of hearing on the mastoid to that obtained by way of the vertex or forehead, was about as three to two. In those cases where bone conduction is longer than air, the Rinne is designated as being “negative.” The lower the pitch of the fork employed, the greater the tendency towards a negative Rinne, and the higher the fork the more likely is the Rinne to be positive. Many otologists employ the c-1 (256 double vibrations) fork or the c-2 fork (512 double vibrations), and some of us prefer the Bezold unweighted a-1 (435 double vibrations) fork.

There are seven or eight varieties of the Rinne reaction. Two forms of the positive Rinne, namely: (1) Air conduction greatly exceeding bone conduction, as in the normal Rinne. (2) Positive Rinne with air and bone conduction considerably diminished as compared with the normal, found in inner ear disease.

Three forms of the negative Rinne: (1) Bone conduction much increased, air moderately diminished, as found in the

ordinary middle ear disease. (2) Bone conduction longer than air but both shorter than normal, so often noted in combined middle and inner ear disease. (3) "Infinitely negative" Rinne when the fork is not heard at all by air, but is heard for a short time by bone. This is very often found in far advanced inner ear disease. The bone conduction factor is probably due to transmission of sounds through the skull to the opposite or hearing ear.

Two forms of the so-called indifferent or plus-minus Rinne: (1) Air and bone conduction of equal length, because air is slightly diminished and the bone conduction slightly increased. This usually means that a slight middle ear affection is present and the hearing is quite good. (2) Air and bone conduction equal in duration but both much shorter than normal; there is usually inner ear disease present and the hearing is very poor. It seems to me that the Rinne is the most valuable of the tuning fork tests, as it gives us an idea of the relation of bone to air conduction and thus furnishes a most important clew to the diagnosis and the location of the hearing impairment—that is, whether the lesion is in the conduction or perceptive apparatus or both.

A good many theoretical objections have been offered to the Rinne test on the ground that, as it is usually performed, bone conduction is tested by means of the longitudinal vibrations of the fork, while air conduction is determined by means of the transverse vibrations of the prongs or tines. However, since the same error is made in every case, the results may be compared with one another, and that suffices for the practitioner, even if it does not for the physicist. In order to overcome these often reiterated objections, Dr. John A. Minton and myself made some experiments and modified the Rinne test, so that the stem of the fork may be used for the testing of both air and bone conduction. After exciting the fork in the usual manner and placing it upon the mastoid until it is no longer heard by bone, the end of the stem is inserted into rubber tubing, which has at its other end a hard rubber olive shaped perforated tip, which is inserted into the ear. The vibrations of the stem are thus conducted along the lumen of the tube to the ear by air conduction. Physical experiments show that the sound waves are hardly if at all transmitted through

the wall of the tubing, but are conducted by the air column in the lumen. Thus it is very easy to use the stem of the fork for both air and bone conduction, and the various objections, we believe, fall to the ground.

The dimensions of the tubing used was 51 cm. length, width of lumen 0.7 cm. and thickness of walls 1.5 mm. In a very brief communication Prof. Bruehl a good many years ago suggested a somewhat similar procedure, but did not base his conclusions on the physical experiments which we made. This report we were able to find only recently.

Gellé Test.—This is used for determining the mobility of the footplate of the stapes. A Politzer or other bag is connected by way of tubing and an olive shaped tip with the external auditory meatus. It is essential that the earpiece be so inserted that it is air tight. With the vibrating tuning fork resting on the bulb, vertex or mastoid, the Politzer bag or bulb is compressed. Normally we have a positive Gellé, in that the hearing of the fork is diminished while the air is compressed in the external auditory meatus. In this manner the footplate of the stapes is pushed further into the oval window and the hearing is impaired for the time being. When the Gellé is negative, which is the abnormal reaction, the hearing is not diminished by the compression of the air, because the footplate of the stapes is fixed and cannot be moved further towards the labyrinth.

The fallacies of this test are: (1) That the patients often do not thoroughly understand what is required of them, or (2) do not pay close attention to the fact that the hearing is somewhat diminished during the time that the bulb is compressed in the normal cases, and (3) it often happens that the earpiece does not fit tightly into the meatus, so that there is an escape of air, and it is then impossible to state whether the footplate is movable or not. If, however, there is no definite change in the hearing with compression of the air column, it may give valuable information. This is especially true in cases of typical otosclerosis with marked fixation of the stapes footplate.

Stenger Test for Determining Simulation of Total Unilateral Deafness.—It is a well known fact that when the two ears are simultaneously exposed to two sounds of the same pitch and

intensity, the one that is produced closer to one ear drowns out or masks, so to speak, the sound that is entering the other ear. That is to say, if a sound is produced at a certain distance from the right ear the individual does not know whether there is a similar sound occurring at a greater distance from the left ear (and therefore apparently of less intensity) than in the right ear. It is essential that a pair of forks be employed of exactly the same pitch, as it is also best that the patient be blindfolded so that he does not know that two forks are being used at the same time.

We usually employ (following Stenger's suggestion) a pair of Bezold's a-1 (435 d. v.) forks. Having struck both forks, one of them is held a certain distance from the left ear, for instance, ten inches. In the normal individual if a fork of the same pitch is placed less than ten inches from the opposite ear, for instance, five inches, he will hear only in the ear at which the fork is a shorter distance, namely, the right one, because the sound coming from that short distance drowns out sound entering the other ear at a distance of ten inches. If, for example, a patient pretends his right ear is deaf, test the left or well ear and find out the distance at which the fork will be heard—let us say, ten inches. In the right ear, which is said to be deaf, the individual will, of course, deny hearing all sounds, no matter how close the fork is approached to that ear. He hears it but denies having any perception of the sound. With one fork kept vibrating, let us say, four inches from the right ear, and the other fork approaches to within eight inches of the left ear, the patient will say that he does not hear any sound at all, even though he previously admitted hearing the fork with the well ear (that is, the left ear) at a distance of ten inches. The reason is this: the fork held in the right ear will drown out the sound of the fork held eight inches from the left ear. In view of the fact that he claims to be deaf in the right ear, he will say that he hears nothing at all, whereas if he really were deaf in the right ear he would not hear the tuning fork held near that ear but would certainly hear the fork vibrating in the vicinity of the left or well ear. Dr. W. A. Wells of Washington recently described a modification of this test which is very simple and ingenious. He uses a piece of rubber tubing of one-quarter inch caliber, 30 inches in length

and of a firmness to give good conduction of sound. In one end of the tube is an earpiece which fits well in the auditory meatus, while into the other end is inserted the stem of a long sounding fork of low pitch (about 120 d. v. per second). After blindfolding, and with the earpiece in the supposedly deaf ear, the vibrating fork is conducted carefully to the meatus of the opposite ear, not too close, but to a point at which it has been previously ascertained that the patient could distinctly hear it; the really deaf person now reports hearing the fork as soon as it comes into the range of hearing of the good ear. The malingerer, unconscious of any hearing in his good ear, because of the lateralization of the sound to the alleged deaf ear (the side of the greater intensity), continues to report no hearing and this gives incontestable proof of willful simulation.

Besides the fundamental tests are others occasionally used, such as the Politzer, for determining the patency of the eustachian tube; the Bing test, for noting whether there is any difference in bone conduction on closing the auditory canal; the Lucae-Dennert test of air conduction, in which it is noted whether the forks are heard louder with or without the closure of the external auditory canal, etc.

Cranial resonance, as described years ago by Politzer, Kessel and many others, has recently been restudied by A. Goldberger, Schoen, Claus and others. The ears of the patient are connected with those of the examiner by means of a double phonendoscope, and the fork placed on the patient's head and then on the examiner's. We have not found that this test gives any more reliable findings than those obtained with the ordinarily performed tuning fork tests.

For testing highest tones, various types of whistles, such as Galton, the Edelman-Galton and the Schaefer-Galton have been devised, as well as the various types of monochords, especially the Schaefer-Struycken. Whistles are supposed to have an upper limit of about 20,000 double vibrations, and it is well to remember that with them one can test only by way of air conduction. The difficulties and fallacies connected with the use of whistles are several. In the first place, it is hard for an individual to distinguish between the blowing sound and the actual high pitched whistle of the high tones. In the second

place, there is difficulty in obtaining uniform pressure of air unless certain devices are employed. Lastly, in the handling of the whistles, unless great care is exercised, rusting or other impairment of the fine edges occurs and causes a decided change in the pitch of the tone. I believe that the Schaefer-Galton possesses certain advantages over the other types, in that:

1. The aperture is fixed at a constant point and thus needs no adjustment.
2. It requires only one hand to manipulate; and,
3. It is considerably cheaper than the Edelmann-Galton whistle.

One of the most reliable instruments for the testing of high tones is the monochord, and the Schaefer-Struycken model is probably the best one for this purpose. The monochord is much more dependable than the whistles and less easily disarranged. By means of it, the high tones can be tested by both air and bone conduction, and it is a fact that with this instrument the very highest tones are better appreciated by bone conduction than by air. The lower tones of the monochord are evoked by transverse vibrations when stroking the wire with a violin bow. The higher tones are longitudinal, and are produced by rubbing the wire with a pad of cotton, etc., moistened with turpentine or alcohol. When bone conduction is being tested, the end of the monochord which has an ivory knob is placed against the mastoid process or some other part of the skull. It is usually assumed that high tones are better heard by air conduction than by bone. With the monochord, however, the very highest tones are better appreciated by bone, as previously stated. Kalaehne says that with the monochord the high tones are better heard by bone because the amplitude is the same for air and bone conduction. With tuning forks this is not possible, because the stem vibrations practically always have an amplitude of only about one one-hundredth of that of the prongs. He believes that with the lowering of the upper tone limit, bone conduction is relatively more diminished than is the air conduction.

Recording of Functional Tests.—There has always been great difficulty in devising a method for recording the tests so that the information therein contained would be readily

accessible to and understood by all readers, as well as to give a reliable index of the findings for the examiner himself. With some slight modifications, we have employed the acoumetric formula adopted at the eighth otologic congress in Buda Pesth in 1909, and have combined it with the diagram long used in the Vienna University Clinic. With either of these methods it is easy in a few moments to record the tests and have all the information available in a small, compact space. Many otologists develop for themselves schemes which seem adequate, but it is highly desirable that some method be designed which would be understood by readers throughout the world.

RECAPITULATION OF PRINCIPAL HEARING TESTS.

1. Observation of the patient.
Loudness of voice (usually loud in severe inner ear disease; usually low in marked middle ear disease).
Close attention and evident lip reading on part of patient, with often appearance of anxiety in effort to hear.
2. Otoscopic examination.
Inspection of:
 1. Auricle.
 2. External auditory meatus.
 3. Tympanic membrane.
 4. Mucosa of tympanic cavity, if perforation of drum membrane is present.
 5. Mastoid region.
3. Nasal, nasopharyngeal and pharyngeal examination.
4. Testing with speech.
With patient's eyes closed or averted and opposite ear closed, use:
 - a. Unaccentuated conversation or whisper, employing high and low pitched numbers or words and combinations of high and low pitched sounds.
Designate in feet or meters or subdivisions thereof the distances heard, or state if ad concham, or not at all. If unaccentuated whisper is heard 1 or 2 meters need not use conversation.
5. Inflation with Politzer bag or by catheter, and again test hearing with speech (whisper or conversation).

6. Tuning fork tests.
Range of hearing:
Lower limits from C-2 (16 d. v.) upwards—i. e., C-1, C, c, c-1, c-2, c-3, c-4, c-5 (4,096 d. v.).
Upper limits: a. Higher forks, c-4 (2,048 d. v.) and c-5 (4,096 d. v.).
b. Galton whistle, preferably Edelmnn-Galton whistle or Schaefer-Galton.
c. Monochord.
1. Weber test for lateralization. Fork placed on median line of vertex, forehead or root of nose.
 - a. Normally heard in vertex ("in the head").
 - b. Usually lateralized in worse hearing ear in conduction apparatus impairment. If both ears have middle ear affection sound goes to worse of the two ears.
 - c. Usually lateralized in the better ear if disease of perception apparatus is present in other ear. If both ears have perception impairment, sound usually lateralized in the better ear.
2. Schwabach test for duration of bone conduction in the individual as compared with the normal, using living control, or comparing with average hearing for the particular fork employed. Fork usually placed on median line of vertex or forehead; it may be set on mastoids. Note whether bone conduction is normal, lengthened or shortened, a slight diminution being of no significance. The age of the patient, the thickness of the hair or bones, the manner of application of the fork, firmness of contact of fork, etc., may give variation in length of bone conduction. A definite lengthening of bone conduction means impairment of conduction apparatus (adhesions, fixation of stapes, etc.). A definite shortening of bone conduction means involvement of the perception mechanism (inner ear or auditory nerve). A decided change in bone conduction is in many ways the key to diagnosis and prognosis in ear disease.
3. Rinne test for comparison of air with bone conduction in the same individual. The stem of the fork is placed on the mastoid (avoiding contact with auricle), and when no

longer heard the prongs are held close to without touching auricle or vibrissæ, and parallel with the ear, and the duration of hearing by air noted.

If a negative Rinne is suspected—e. g., if the Schwabach was found lengthened, test air conduction first and then bone conduction.

Normally the Rinne is positive (air conduction longer than bone). There are about seven varieties of Rinne (two forms of the positive, three varieties of the negative and two of the indefinite or plus-minus type.)

4. Gellé test, for determining mobility of the footplate of the stapes. Compressing the air in the external auditory meatus gives diminution of hearing in normal cases by pushing stapes into oval window; where fixation of stapedial footplate is present, no change in hearing occurs with increase in air pressure.
5. Stenger test, for unmasking simulation of total unilateral deafness. Two forks of exactly the same pitch are used; patient unaware that more than one fork is sounding. The fork nearer one ear drowns out sound of fork at other ear.
6. Audiometers are being used considerably in testing, and with them we get accurate measurements of hearing in sensation units. Audiograms obtained give graphic idea of the state of hearing, which may be preserved for future reference and comparison. With properly calibrated forks, provided with the "constant" of damping or decrement, one is able to obtain a similar curve, but if many pitches are to be tested the process is somewhat more tedious than with the audiometer.
7. Resonators and other appliances should also be employed as previously mentioned in the methods of functional testing of hearing.

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XXVI.

STUDIES IN OTITIC SEPSIS.

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Much has been learned of otitic sepsis during the past decade. The diagnosis and management of cases of septic sinus thrombosis and lateral sinus phlebitis have to a certain extent become standardized. However, in the light of a wider experience the accepted standards of diagnosis and the indications for surgical therapy may be modified to some degree. It is now possible to determine the nature of a given case more exactly and to apply a given surgical procedure more specifically and with more assurance of a successful outcome than was heretofore possible. Furthermore, the wider the experience with cases of this sort, the less do they appear to fall within set standards either for diagnosis or for treatment; and our riper knowledge permits an individualization of each case that was formerly impossible.

In otitic infections, proper evaluation of the component parts of the clinical picture inevitably leads to an earlier detection and eradication of the sinus phlebitis producing the otitic sepsis. Where there is an improper interpretation and correlation of the laboratory and clinical findings, failure to establish early diagnosis results. The twelve cases reported herewith illustrate these principles. They are cases which occurred in the ordinary course of practice and were not selected for any unusual features presented.

The ensuing paper is divided into two sections, the first comprising the case reports and the second a discussion based on the clinical material recorded.

PART I.

CASE REPORTS.

Case 1.—A female, age 50 years, was admitted to the United Israel-Zion Hospital on November 11, 1925. Three weeks previously she had had severe pain in the right ear which soon subsided. There was no aural discharge at the time. Three days before admission the pain in the right ear recurred and the drum ruptured spontaneously with a resultant purulent otorrhea. The patient had very little pain at the time of admission. The temperature was 101 F. Otoscopic examination showed a marginal perforation in the right drum and a moderate discharge in the middle ear. A roentgenogram of the right mastoid process showed sclerosis and evidences of acute destruction interspersed.

Urinalysis revealed sugar to the extent of 2.1 per cent. The blood sugar was 280 mgms. per cc. The blood count was as follows: R. B. C., 4,020,000; Hb., 80 per cent; W. B. C., 9,000; polys, 81 per cent.

The patient was given antidiabetic treatment and was under observation until December 14, 1925. At that time her urine became sugar free, but the blood sugar was still high, 250 mgms. per cc. While the local condition in the ear did not progress, it showed no tendency to improve, and operation on the mastoid was advised.

A simple mastoidectomy was performed on December 15, 1925. A sclerosis of the mastoid was found in which were necrotic areas leading toward the tip and toward the sinus. The sinus plate was found eroded, and when it was removed a small area of the outer sinus wall was found covered with granulations.

During the following week the patient complained of severe pain in the frontal area and headache over the right side of the head. The temperature gradually rose, reaching 104 F. on the 22nd. Blood counts taken daily showed no change in the number of red cells and hemoglobin till the seventh count, taken on the 21st. This showed a drop in the hemoglobin to 70 per cent. The total white count showed a gradual increase, reaching 19,600, with 92 per cent polys on the 22nd. A blood culture taken on the 21st proved negative.

The patient was reoperated upon on the 22nd of December. The sigmoid sinus was fully exposed, from the knee to the bulb, and plugged off above and below with iodoform gauze. The sinus was incised and was found to contain free pus. Free bleeding was obtained from above and below, and the sinus was then obliterated. The internal jugular vein was next ligated.

The patient was transfused immediately after operation with 300 cc. of whole blood. The temperature returned to normal within two days and remained so until the time of discharge from the hospital, February 15, 1926, at which time the ear condition was entirely cured.

Summary.—This case presents

1. The presence of diabetes mellitus.
2. An asymptomatic course between the onset of the otitis and the first operation, a period of five weeks.
3. An involvement of the sinus wall at the first operation.
4. A negative blood culture.
5. The presence of fluid pus in the sinus.
6. The drop in hemoglobin and the rise in the white count.
7. The presence of unilateral headache.

Case 2.—A male child, age 6 years, had had a right mastoidectomy performed at the United Israel-Zion Hospital on October 8, 1927. A perisinal abscess was found at the time. He was discharged from the hospital in seemingly good health on October 17th. On the 18th, the temperature rose to 102 F. and on the following day to 103 F. The child complained of severe pain over the right side of the head and was admitted to the New York Polyclinic Hospital. The temperature for the following two days was low, having dropped to 100 F. The blood culture was negative. The blood count was as follows: R. B. C., 3,800,000; Hb., 65 per cent; W. B. C., 19,000; polys, 86 per cent. The principal complaint was headache.

General medical examination was negative. Repeated blood counts—three—showed a persistence of the polynucleosis and the diminution in red cells and hemoglobin. The headache was likewise persistent. A diagnosis of sinus thrombosis was then made on the symptoms of headache, increased polynuclear count, lowered hemoglobin percentage and a history of a peri-

sinal abscess at the first operation. The temperature remained below 100 F.

On October 21st, the mastoid wound was reopened and the sinus exposed from the knee downward toward the bulb. The granulations were curetted from the sinus wall. It was then plugged off above and below and incised between. Removal of both upper and lower plugs revealed no bleeding. Clots were removed from the bulb and from the torcular end until free bleeding was established. The sinus was then obliterated. The internal jugular vein was not operated upon.

The child's temperature returned to normal on the 22nd and remained so until he was discharged from the hospital. He was given a transfusion of whole blood, 300 cc., on the 22nd of October. On November 1, 1927, the mastoid wound was sutured and the child discharged from the hospital on the 8th of November. He has fully recovered.

Summary.—This case presents

1. A perisinal abscess at the primary operation.
2. Ten days after operation, a rise in temperature, unilateral headache, increased polynucleosis, which was persistent, and a low hemoglobin and red cell count.
3. The preoperative diagnosis of sinus thrombosis on the factors listed in No. 2, in spite of a low temperature.
4. A negative blood culture.
5. The resultant cure without obliteration of the internal jugular vein.

Case 3.—Female child, age 7 years, was admitted to the Beth Israel Hospital on February 20, 1927. Two weeks before admission the child complained of pain in the right ear. The pain soon abated but returned with increased severity one week later. For the following week her temperature was 103 F., and she had two chills. A paracentesis was performed a few hours before admission and pus was obtained.

On admission the temperature was 103.2 F. The drum was red, and through the incision in it a pulsating discharge of pus was noted. There were no signs of drooping of the postero-superior canal wall. The mastoid process was tender on pressure. The blood count was as follows: R. B. C., 3,650,000; Hb., 70 per cent; W. B. C., 17,200; polys, 80 per cent. Blood culture yielded streptococcus hemolyticus, three colonies to the plate.

A simple mastoid operation was performed on February 20, 1927. An acute coalescent mastoiditis was found. The sinus plate was eroded and a large perisinal and epidural abscess was found, the latter in the region of Trautmann's triangle. The sinus was discolored and appeared white in spots. It was fully exposed, and on incision a disintegrated thrombus was found. It was removed and free bleeding was obtained from both ends. The sinus was then obliterated. The internal jugular vein was not operated upon.

The temperature returned to normal and remained so until the time of discharge from the hospital. A second blood culture, taken on the 22nd of February, proved sterile. The blood counts taken daily until the 26th of February showed a gradual decline in the total white and polynuclear count until normal was reached on the 26th. The red cells and the hemoglobin percentage, however, remained at the level noted before operation. A transfusion of 300 cc. of whole blood was therefore given. The red cell count and the hemoglobin then mounted, and, at the time of discharge, March 11, 1927, the red cells numbered 4,500,000 and the hemoglobin was 80 per cent. The child has fully recovered.

Summary.—This case presents

1. A general sepsis at the time of admission to the hospital.
2. A positive blood culture.
3. The presence of a thrombus in the sinus at operation.
4. A decreased hemoglobin percentage and reduced number of red cells, and their rise to normal limits after operation.
5. The resultant cure without obliteration of the internal jugular vein.

Case 4.—Male, age 18 years, was admitted to the Beth Israel Hospital on January 22, 1927. One week prior to admission he complained of a sudden pain in the left ear and a discharge of brownish pus from that ear. He complained constantly of a persistent dull headache over the left side of the head. On the third and fourth days of his illness, he vomited several times. His temperature during the entire week ranged between 102 F. and 104 F.

On admission to the hospital, his temperature was 103 F. There was a marked swelling over the left mastoid process. The otoscopic picture presented a profuse pulsating discharge

under pressure, a sagging of the posterosuperior canal wall and a narrowing of the canal lumen. The blood count was as follows: R. B. C., 4,000,000; Hb., 80 per cent; W. B. C., 18,000; polys, 87 per cent. A blood culture was taken and was reported sterile the following day. Culture of the aural discharge yielded streptococcus hemolyticus.

The patient was operated upon the day after admission. A left simple mastoidectomy was performed. The swelling over the mastoid process was due to edema. A coalescent mastoiditis was found and a large perisinal abscess, which also involved part of the dura in the region of Trautmann's triangle, was uncovered. The sinus wall was found collapsed and green in color. The simple mastoid operation was completed, and the sigmoid sinus then fully exposed from the knee downward toward the bulb. It was blocked off above and below and incised between the obliterating gauze plugs. Fluid pus and pieces of disintegrated thrombi were removed by suction until free bleeding was established from above and below. The internal jugular vein was then ligated.

For the next two days the temperature ranged between 102 F. and 106 F. Another blood culture taken on the 26th was sterile. The cerebrospinal fluid was examined on the same date and was normal in pressure, chemistry and cytology. The eye grounds showed only a slight congestion of both retinal fields. On the 27th, the patient had a chill and a rise in temperature to 106 F. During this entire postoperative period repeated blood counts showed no variations in the total red cell and white cell counts and the hemoglobin percentage: the findings were the same as those noted before operation. The percentage of polynuclears, however, had showed a gradual drop after operation from 87 to 67 per cent.

The temperature then dropped to reach 100 F. on the 29th, but, following removal of the sinus plugs on the following day, rose to 104 F. For the following week the temperature ranged between 100 and 101 F., returning to normal during the third week. Repeated blood counts during this period showed a gradual drop in the total leucocytosis until normal (9,400) was reached on the 8th of February. The red cell count and the hemoglobin percentage always remained at the normal level noted prior to operation. The only complaint made by the pa-

tient was persistent headache, which did not subside until the third week after operation. The patient was discharged from the hospital on the 15th day of February. The subsequent dressings were done away from the hospital.

Summary.—This case presents

1. A virulent infection.
2. Edema over the mastoid process (Greisinger's sign).
3. The presence of fluid pus in the sinus.
4. Repeated negative blood cultures.
5. Persistent headache.
6. Persistence of postoperative septic temperature, while the reduction in the number of polynuclears spoke for a subsidence of the infection and the maintenance of the high total white count showed a strong resistance on the part of the patient.

Case 5.—Male child, 13 years, was admitted to the United Israel-Zion Hospital on July 28, 1927. Following sea bathing two weeks prior to admission, the child complained of pain in the left ear. A myringotomy was performed and pus was obtained. The child then felt well until the 25th of July, when the pain in the left ear became intense and the temperature suddenly rose to 103.5 F. This condition persisted until admission.

On admission the temperature was 105.8 F. The blood count was as follows: R. B. C., 4,100,000; Hb., 80 per cent; W. B. C., 19,370; polys, 89 per cent. Blood culture taken at this time proved sterile.

A simple mastoidectomy was performed four hours after admission to the hospital. The entire mastoid process was coalesced. There was a large perisinal abscess with granulations on the sinus wall. The sinus was exposed throughout its entire extent in the mastoid process and was then opened. A large clot was found in its interior. This was removed and free bleeding established. The sinus was then obliterated and the internal jugular vein ligated. Culture of the pus from the mastoid and from the thrombus yielded a nonhemolytic streptococcus.

The temperature returned to normal on the next day. The child then had a chill and a rise in temperature to 106 F. on the 30th of July. The temperature then dropped to normal and remained so until the time of discharge from the hospital, August 7, 1927.

Summary.—This case presents

1. Sudden onset of fever and severe pain two weeks after the appearance of the acute otitis media.

2. A perisinal abscess and granulations on the sinus.

3. A negative blood culture.

4. A formed thrombus within the sinus.

Case 6.—Male, age 16 years, was admitted to the United Israel-Zion Hospital on February 17, 1926. He had had diphtheria at the age of one year, followed by a persistent discharge from both ears. On February 13th, he began to complain of severe pain in the left ear, frontal headache, dizziness and intermittent fever.

On admission to the hospital the temperature was normal. There was a pulsating discharge from the left ear, and granulations were present in the middle ear. There was a large marginal defect in the drum. Functional examination of the labyrinth revealed normal responses to all tests. Sound was lateralized to the left ear. The patient heard the shouted voice with the left ear when the noise apparatus was in the right ear; but for practical use hearing was destroyed. The blood culture was negative. Blood count gave the following: R. B. C., 3,900,000; Hb., 75 per cent; W. B. C., 14,300; polys, 80 per cent.

On February 19th the patient had a severe chill, with a rise in temperature to 105.8 F. He again had a chill on the 20th. A second blood culture proved negative.

A radical mastoidectomy was performed on the left ear on February 20th. A sclerotic mastoid was found in addition to a cholesteatoma which filled the entire middle ear and attic. The facial nerve was found exposed in its horizontal portion. Working toward the sinus, there was a gush of pus which came from a large perisinal abscess. The sinus was exposed and opened. Fluid pus was present in the interior. Free bleeding was established from above but not from the bulbar end. The sinus was obliterated and the internal jugular vein ligated.

The temperature returned to normal on the second day after operation and the patient made an uneventful recovery. He was discharged from the hospital on March 17, 1927.

Summary.--This case presents

1. A cholesteatoma.
2. Acute onset of symptoms in presence of a chronic lesion. The headache, intermittent fever and dizziness pointed to some intracranial lesion.
3. The two negative blood cultures.
4. Fluid pus in the sinus.

Case 7.—Female child, age 7 years, had had a left simple mastoidectomy in 1922. In December, 1924, she had a recurrence of her mastoid infection following an attack of scarlet fever and was reoperated upon. On April 30th, after having completely recovered from her condition, she suddenly developed high fever and a discharge from the left ear. Several petechial hemorrhages made their appearance over the abdomen and in the conjunctivæ.

The child was admitted to the United Israel-Zion Hospital on May 3, 1925. Blood culture, on admission, showed 400 colonies of streptococcus hemolyticus to the cc. The blood count was as follows: R. B. C., 3,900,000; Hb., 65 per cent; W. B. C., 20,200; polys, 88 per cent. The temperature was 104.

The child was operated upon the day of admission. Nature's attempt at forming a new cortex was evident. A fistula was present through the squamomastoid suture, through which pus came away under pressure. The mastoid operation was revised and the sigmoid sinus was found exposed. Its wall was thick and white in appearance. A formed thrombus was removed from its interior and free bleeding established from the upper and lower ends. The sinus was then obliterated. The internal jugular vein was ligated.

Postoperatively the temperature varied daily between 102 and 105, with frequent chills. A blood transfusion (250 cc. of whole blood) was given on May 4th. New petechiæ appeared over various parts of the body. On May 9th the spleen was felt three fingers below the free border of the ribs. On May 14th an abscess of the left ankle and the right index finger was noted. The following day a right lobar pneumonia was evident. Signs typical of a peritonitis set in on the 16th, and the child died on May 19th.

The blood count remained the same throughout the course of the disease. The second blood culture, taken on May 11th, showed four colonies of streptococcus hemolyticus to the plate.

Summary.—This case presents

1. Repeated infections of the mastoid process.
2. The finding of an exposed sinus.
3. The virulence of the infection and its apparent selection of the viscera.
4. The positive blood culture.
5. The decrease in the number of colonies after operation, with the secondary lesions eventually causing death.

Case 8.—Male child, age 11 years, was admitted to the United Israel-Zion Hospital on February 1, 1927. Eight days prior to admission the child developed severe pain in the right ear and a high temperature. The right drum was incised and pus obtained. The fever persisted, nevertheless (between 104 and 105, according to the parents), and the discharge from the ear became profuse. On the day of admission the child had a severe chill.

Temperature on admission was 105.2. Otoscopic examination showed a profuse pulsating discharge from the right middle ear. There was no sagging of the canal wall nor any narrowing of the canal lumen. The right mastoid process was extremely tender. Blood count: R. B. C., 3,800,000; Hb., 65 per cent; W. B. C., 18,600; polys, 90 per cent.

A simple mastoidectomy was performed on the day of admission. A hemorrhagic mastoiditis was found. There was no free pus and no breaking down of the cell walls. The sinus and dura of the middle cranial fossa were exposed and found normal in appearance.

For the following week, the temperature ranged daily between 101 and 106 with daily chills. Two blood cultures taken were negative. An examination of the spinal fluid on the 3rd of February gave normal findings. On the 5th, the spleen was found to have enlarged two fingers below the costal margin. Widal was negative.

On the 8th of February, the sigmoid sinus was opened and found to contain an organized thrombus, which, when cultured, yielded a streptococcus hemolyticus. The internal jugular was then ligated and the sinus obliterated.

During this entire time the blood count showed the same picture as at the time of admission. The temperature during the following three weeks dropped by lysis to 100. On March 1st a swelling of the glands on the right side of the neck was noted. The temperature thereafter fluctuated between 101 and 103 until the 19th of March, when complete softening of the glands was noted and incision and drainage of the infected glands performed. The temperature then returned to normal. The child was discharged from the hospital, completely recovered, on the 20th of April, 1926.

The blood count in this case always showed the presence of increased leucocytosis, both relative and actual. The hemoglobin never dropped below 65 per cent.

Summary.—This case presents

1. The typical clinical picture of an acute hemorrhagic mastoiditis.
2. Normal appearance of the sinus wall.
3. Continuance of the sepsis after the simple mastoid operation.
4. Negative blood culture.
5. The presence of a formed thrombus in the sinus.

Case 9.—Female child, age 6 years, was admitted to the United Israel-Zion Hospital on April 1, 1925. Two weeks prior to admission she had had a right simple mastoidectomy performed at the Jewish Hospital in Brooklyn. At that time a large perisinal abscess was found at operation. At the time of her first dressing, ten days before admission to the United Israel-Zion, a severe hemorrhage occurred during the removal of the packing from the mastoid wound. The wound was re-packed at that time, but, when dressed the following day, a hemorrhage again occurred. On the day of admission the blood culture yielded streptococcus hemolyticus, 13 colonies to the plate. The temperature was 104.

The child was reoperated upon April 1st. The mastoid wound was reopened. The sigmoid sinus was exposed above and below the site of the previous exposure, and plugs of iodoform gauze were inserted to obliterate the lumen. On removal of the upper plug, free bleeding followed. The bulb, however, contained a thrombus, which was removed by suc-

tion. Sluggish bleeding followed this procedure. The internal jugular vein was then ligated.

The child was transfused with 300 cc. of whole blood on the 5th. The temperature postoperatively was septic, ranging from normal to 104 daily until the 30th of April. The red cell count and hemoglobin remained at a normal level (four to five million of 80 per cent hemoglobin). The white cell count during this period averaged 13,000 with 84 per cent polys. On April 12th she developed a swelling and a fixation of the right hip. On the 19th, a swelling appeared over the right leg and right forearm. On the 20th, a fluctuating swelling of the left arm was noted. On the last date, incision and drainage of the abscesses of the arms and left leg were performed.

From April 30th to May 5th the temperature varied between normal and 102 F. On May 5th, fluctuation was noted in the swelling over the right hip. The roentgen plates showed no apparent involvement of the hip joint at this time. This swelling was incised and pus evacuated.

Repeated X-ray examinations made during the month of May showed a progressive destructive arthritis of the right hip joint. This was accordingly treated by the orthopedist. Several subsequent incisions of the hip joint were made before complete healing resulted.

From then on until the time of discharge from the hospital, July 7th, the temperature varied between normal and 100, with an occasional rise to 102. All blood cultures after the first were sterile. The otitic lesion had completely healed at the time of discharge, but the arthritis still required treatment at the hands of the orthopedist.

Summary.—This case presented

1. The presence of a perisinal abscess.
2. The rupture of the sinus during the dressing.
3. The presence of a thrombus within the sinus.
4. The positive blood culture.
5. The metastatic lesions.
6. The stormy clinical course.

Case 10.—A boy, age 7 years, was admitted to the Beth Israel Hospital on March 27, 1926. He had had a lobar pneumonia three weeks prior to admission. On March 13th, he complained of pain in both ears. The following day both ear drums

were incised and the middle ears continued to discharge pus up to the time of admission.

On the day of admission the temperature was 101. Both ears presented a profuse discharge from the middle ear, under pressure, and a narrowing of the canal lumina. The mastoid processes were tender on pressure. The blood count was as follows: R. B. C., 5,800,000; Hb., 80 per cent; W. B. C., 13,240; polys, 76 per cent.

A bilateral simple mastoidectomy was performed on the day of admission. A coalescence of the cells and fluid pus was present in both mastoids. The sinus plate on both sides was diseased and showed evidences of erosion on the right side. The outer walls of the sinuses were normal in appearance. The wounds were packed with iodoform gauze.

For the following four days the temperature stayed below 101.5. The wounds were dressed on March 31st. On removing the packing from the right wound, a gush of blood followed. The wound was immediately repacked. When the child was dressed again, on the 3rd of April, a similar occurrence followed removal of the packing from the right mastoid wound. The bleeding at this time was controlled by a plug of iodoform gauze inserted into the opening in the sinus plate. The temperature immediately rose to 105 and gradually dropped to normal during the next five days. Blood culture was sterile. The blood count, however, showed a drop in the number of red cells to 3,500,000, and a drop in the hemoglobin to 65 per cent. The total white count was 8,000, polys 75 per cent. From the 8th to the 16th of April, the temperature varied between 100 and 103. On the last date the child complained of pain in the right hip and had a chill with a rise in temperature to 104.3. A blood culture taken on the 14th and one taken on the 16th were both sterile. The red blood cells had further dropped to 3,000,000 and the hemoglobin to 55 per cent.

The right mastoid wound was reopened on the 16th of April and the sigmoid sinus exposed on either side of the previous defect. The plug was removed from the site of the old exposure. On releasing the pressure of the upper and lower plugs, free bleeding was obtained from the distal and proximal ends of the sinus. No thrombi were demonstrable in the bulb. The internal jugular vein was then isolated and ligated.

The temperature ranged between 101 and 104 until the 21st. The child was then transfused with 250 cc. of whole blood. On this day the blood count showed the following: R. B. C., 4,900,000; Hb., 70 per cent; W. B. C., 9,500; polys, 64 per cent. A second transfusion was given on the 26th. The hip condition was treated by Dr. Harold Lusskin, who diagnosed it as an acute arthritis of the right hip joint. It cleared up under traction and abduction. The child was discharged from the hospital on May 22nd, fully recovered.

Summary.—This case presents

1. The normal appearance of the sinus walls at the time of the first operation, except for an erosion of the sinus plate on the right side.
2. The rupture of the right lateral sinus during the dressing.
3. The subsequent development of sepsis.
4. The negative blood cultures.
5. The metastatic hip lesion.
6. The drop in the hemoglobin and red cells during the development of the sepsis.
7. The absence of a thrombus in the sinus.

Case 11.—Male, age 30 years, was admitted to the Beth Israel Hospital on November 25, 1927. The patient had had a chronic otorrhea from the left ear for six years. This followed bathing. Following an attack of acute follicular tonsillitis one month before admission, the patient began to complain of a dull pain in the ear. Examination prior to admission to the hospital showed a large marginal defect in the left drum. The middle ear was filled with cholesteatomatous masses. The labyrinthine function was normal. The cochlear function was impaired for useful hearing: the audiogram showed a 79 per cent average loss. The roentgen plate showed a sclerosis with a large clear area due to a cholesteatoma.

Temperature on admission was 101. The blood count showed the following: R. B. C., 5,000,000; Hb., 90 per cent; W. B. C., 15,000; polys, 83 per cent. Culture from the left ear showed a staphylococcus aureus.

The usual radical mastoid operation was performed on the 26th of November. A large cholesteatoma was found to fill the mastoid process and the middle ear. The lateral sinus was

exposed at the knee during the procedure. The postoperative course was uneventful.

On the 9th of December the patient was taken to the operating room in order to undergo the plastic operation on the canal and have the mastoid wound closed. However, at the onset of the operation, before any instrumentation was begun, the sinus ruptured spontaneously. This was plugged off and the plastic operation was performed on the canal after Panse's technic.

On the 11th of December, the temperature rose suddenly to 104. The hemoglobin dropped to 75 per cent and the white cells increased to 15,250, with 85 per cent polys. A blood culture on that date showed streptococcus hemolyticus, 24 colonies per plate, two days later. The temperature continued high for the next two days.

On December 13th the internal jugular vein was first ligated. The sinus was then exposed above and below the torn portion at the knee and the interior of the sinus was opened. No clot was found, and free bleeding was obtained from the proximal and distal ends of the incised sinus. The sinus was then obliterated.

Subsequent blood cultures were negative. The blood count rapidly returned to normal. On December 20th the posterior wound was closed, and the patient was discharged from the hospital January 7, 1928. At this writing he is still under treatment for the mastoid wound, which is undergoing epidermization under ambulatory treatment and observation.

Summary.—This case presents

1. A cholesteatoma, with an acute infection superimposed.
2. Spontaneous rupture of the sinus.
3. A sudden onset of high temperature two days after rupture of the sinus.
4. Positive blood culture.
5. No thrombus in sinus.
6. The drop in hemoglobin.

Case 12.—Female child, age 7 years, was admitted to the Brownsville and East New York Hospital on January 20, 1927. Two weeks before admission, the child had had a follicular tonsillitis followed by a swelling of the submaxillary glands. The family physician made a diagnosis of mumps at the time. Three

days before admission, the child developed pain in the right ear, which ruptured spontaneously. The child vomited twice before admission.

On admission the temperature was 106.5. The child was in a semicomatose condition. A bilateral Babinski and Kernig were present. There was a paresis of the left internal rectus muscle. The drum showed a small perforation in the inferior quadrant with but a scant discharge of pus. The landmarks were all present. There was no evidence of any mastoid involvement. Blood count showed the following: R. B. C., 3,750,000; Hb., 68 per cent; W. B. C., 13,800; polys, 80 per cent. Blood culture was sterile. Cerebrospinal fluid was normal. There was no evidence of any abdominal involvement. There were no signs of any chest involvement. Widal was negative.

In view of the negative findings elsewhere in the body, both the pediatrician and the neurologist demanded an exploratory operation on the mastoid in spite of my insistence that no mastoiditis existed. A simple mastoidectomy was accordingly undertaken on January 21st. A perfectly normal mastoid was found; the cells were intact. There was no evidence of findings suggestive of a hemorrhagic mastoiditis.

The child continued to run a septic temperature for the following thirteen days, varying between 100 and 104 and 105 daily. Two subsequent blood cultures taken during this period were negative. Repeated Widal examinations and spinal fluid examinations were negative. The blood count the day following operation and for this period showed a drop in the red cells to 2,800,000, with the hemoglobin at 59 per cent. There was a leucopenia present throughout, the total cells never being higher than 7,200, and at one time dropping to 5,600. The polynuclears ranged between 54 and 78 per cent. A smear for malaria was negative. The neurologic findings at the time of admission gradually disappeared, but the child showed the evidences of a prolonged sepsis in that she lost weight and began to look drawn and septic.

On February 5th the child had a severe chill, followed by a rise in temperature to 105. The following day the temperature was normal. On the 7th she had another chill and a rise in temperature to 104. The blood count still showed a leuko-

penia. A blood culture taken on the 6th showed a growth of streptococcus hemolyticus, 40 colonies to the cc.

A diagnosis of primary bulb thrombosis was then made and the child was operated upon February 7th. The mastoid wound was reopened and the sinus fully exposed. It was plugged off above and below and incised between. On removal on the upper plug, free bleeding occurred. On removal of the lower plug no bleeding resulted. A large clot was removed from the bulb. This had partly extended upward into the lower portion of the sigmoid sinus. The sinus was obliterated and the internal jugular vein was resected, beginning at the facial and extending downward for about two inches.

A transfusion of whole blood, 300 cc., was given the following day. The temperature then fell by lysis, and by the 13th of February had reached normal, where it remained until the child was discharged from the hospital. The subsequent blood cultures were sterile. The blood count still showed a leukopenia at the time of discharge, February 21, 1927, at which time she had fully recovered. The wound only required further dressings.

Summary.—This case presents

1. Mumps.
2. Onset of otitis with symptoms of meningeal irritation.
3. Normal mastoid at operation.
4. Negative blood cultures for 21 days after onset of disease, in spite of septic picture.
5. Gradual fall in red cells and hemoglobin.
6. The presence of a persistent leukopenia.
7. The subsequent appearance of a positive blood culture.
8. The presence of a primary thrombus in the bulb.

PART II.

COMMENTS.

Features Presented in the Histories.—In case 1 the important factor is the presence of diabetes. It seems almost academic again to discuss the significance of diabetes in connection with the treatment of aural infection; but the fact that this case was permitted to remain under observation for so long a time after admission to the hospital before active surgical intervention justifies my speaking of it. The essential thing to be remembered is that mastoiditis in a diabetic is almost

painless and asymptomatic. Destruction of the mastoid process will proceed to a fatal termination without any pain.

Consequently operative intervention is indicated earlier than in the usual case of acute mastoiditis if one would forestall intracranial invasion. Had the proper emphasis been placed upon the diabetes and not upon the stationary otoscopic picture operation would have been performed much earlier and the sinus thrombosis might have been averted. In a diabetic it is sufficient indication for mastoid surgery if the otoscopic picture shows no improvement within a reasonable time. One should not wait for pain and tenderness of the mastoid, more profuse discharge, sagging of the canal wall, etc. The fact that the otitis does not show signs of resolution is in itself indicative of a destructive lesion. In diabetics, symptoms appear with the involvement or threatened involvement of the endocranium. In this case the first symptom to appear was unilateral headache, which came on after the first operation.

Headache is an important symptom in any otitic infection. In cases 1, 2, 4 and 6 it presented the typical distribution. The headache due to sinus phlebitis is almost always localized to the affected side. It is a dull, persistent pain over the area operated upon which radiates toward the frontal and occipital areas. Before the primary operation on the mastoid it is almost impossible to attribute any significance to the headache which may be present, because of the evident lesion in the mastoid process. After mastoid surgery, however, headache assumes the utmost significance. If due to the mastoid lesion, it should disappear soon after operation. Where it persists and becomes localized to the side operated upon or where, after it has subsided, the headache returns again with equal or greater intensity over the affected side, a sinus phlebitis should be strongly suspected. If this symptom were given its proper value, an earlier recognition of sinus phlebitis would be possible.

Of the two other prominent symptoms presented by the patient, the temperature reaction is more important than the presence or absence of chills. All of the cases presented a rise in temperature concomitant with the onset of the sinus phlebitis. (The character of the temperature will be discussed under a separate heading.) On the other hand, only four of

the cases (3, 8, 10 and 12) had chills. This leads me to state that only when the sinus phlebitis threatens general systemic invasion is a chill present. The local infection of the sinus will in itself rarely produce a chill. Consequently this symptom is less apt to appear early in the course of sinus phlebitis. Temperature, on the other hand, is practically always present with any infection, and consequently should be expected with an infected sinus.

Features in the Clinical Picture.—With the exception of cases 1, 2 and 5, all showed a septic temperature. This is the type of temperature curve that one would expect with a systemic infection. That a septic temperature is not always present in connection with a sinus phlebitis should be borne in mind. What I wish to emphasize in this regard is that the absence of a septic temperature or the presence of only a slight elevation in temperature does not necessarily exclude the diagnosis of sinus phlebitis.

A septic temperature which appears at the onset of an otitis and continues in evidence presents an added significance. It should immediately lead one to suspect the presence of a hemorrhagic mastoiditis. Case 8 illustrates this type of lesion, wherein at no time is there any coalescence of the mastoid cells with gross necrosis and purulency. The cell walls are intact, and microscopically it has been demonstrated that this type of lesion is characterized by a multiplicity of thrombi in the venules of the mucosal lining. Such a case consequently presents an otitic sepsis from the outset of the lesion, and immediate surgery of the mastoid process is indicated to prevent extension of the thrombosis to the lateral sinus. In case 8, the lesion had progressed eight days before the simple mastoidectomy was performed, and subsequent to the operation the septic temperature continued. This should have immediately led one to suspect the involvement of the lateral sinus despite its normal appearance, since, in a hemorrhagic mastoiditis uncomplicated by sinus thrombosis the temperature drops soon after operation.

The otoscopic picture before operation gives no information as to the presence or absence of a sinus phlebitis. The presence of a swelling over the mastoid process in the course of an otitic purulency, however, should be closely investigated.

Where no fluctuation is present and the swelling seems to be situated over the posterior part of the mastoid process rather than close to the auricle, one should suspect an edema of the soft tissues rather than a subperiosteal abscess. The swelling due to edema is called Greisinger's sign. It is always indicative of an interference with the circulation in the vicinity of the mastoid process and is usually due to an interference with the flow of blood through the lateral sinus. Most frequently only a perisinal abscess is found in conjunction with this sign, but the possibility of a subsequent thrombosis or infective phlebitis should be borne in mind.

Signs that come on later in the course of an otitic sepsis are an enlarged spleen, as found in cases 7 and 8, interference with the venous return from the optic nerve and consequent pathology of the nerve head (congestion of the retinal veins, papilledema), petechial hemorrhages and metastatic lesions. As regards papilledema, I have not been able to rely on its presence as a diagnostic aid. Where it was present, the thrombus was of the obliterating type; and in the majority of my cases, including the ones here reported, no ocular fundus change was noted. Even after surgical obliteration of a sinus wherein only a mural clot was found, nothing more than a slight congestion of the retinal vessels was ever noted.

Metastatic lesions may occur early or late in the course of a sinus phlebitis. In case 7, the metastatic abscesses occurred almost concomitant with the onset of the clinical sepsis. In case 9, eleven days elapsed between the operation for obliteration of the sinus and the appearance of the hip lesion. Similarly, in case 10, the metastases occurred late. The location of the secondary lesions are more important in the prognosis of the case than in the diagnosis of the sinus phlebitis. Where the superficial structures alone are involved—and by that I mean skin, muscles and joints—the prognosis for recovery is favorable, because the abscess acts as a fixation abscess and clears the blood stream of the infection. Where the viscera are selected by the organism, the prognosis is unfavorable because of the inability to successfully reach the abscess and evacuate it. Then again, interference with the function of a visceral organ adds to the burden of the patient and further incapacitates him.

Features in the Gross Pathology at Operation.—The findings at operation which would make for a diagnosis of sinus phlebitis differ in the acute and in the chronic cases. In the acute coalescent mastoiditis, the presence of a necrotic sinus plate which, when removed, reveals a normal looking sinus or one that is but slightly discolored, has less significance than the same finding in a case of cholesteatoma or bone necrosis. In the latter instances, the slow progress of the lesion in the mastoid process has produced a gradual destruction of the sinus plate, and the underlying blood vessel is the seat of a chronic inflammatory reaction. One can never estimate the extent of this reaction nor can one judge on the operating table whether the outer coats alone are involved or the interior also. In the acute coalescent mastoiditis the finding of a necrotic sinus plate with no granulations on the sinus wall makes the possibility of a sinus phlebitis unlikely.

A perisinal abscess with granulations present on the sinus wall should be viewed with the utmost suspicion. Almour¹ advises the obliteration of every sinus wherein an extensive periphlebitis is found, regardless of the presence or absence of signs of systemic involvement. He feels that by this procedure one eradicates a potential focus of general sepsis. I am of the same opinion and feel that it is far better to avoid an otitic sepsis than to treat one that has been permitted to develop. In my cases this point is clearly demonstrated. In case 1, had the sinus been obliterated at the first operation, the sepsis would have been stopped in its incipency. Here the perisinal abscess and the extensive granulations on the sinus justified such a procedure. Similarly, in cases 2 and 9, an earlier obliteration of the sinus would have avoided the subsequent sinus thrombosis. In cases 4, 5 and 6, the sinus was obliterated at the time of the first operation on the mastoid process. It is true that in these cases other evidences of systemic infection were present to warrant inspection of the sinus. Nevertheless, I feel that it is possible to avoid the appearance of a general sepsis by eliminating the main factor in its development in the coalescent and chronic mastoiditis—i. e., by removing the infected portion of the sinus wall and obliterating the remainder of the sinus.

In the acute hemorrhagic mastoiditis, no information can be gained by inspection of the sinus wall or sinus plate. In this type of infection a sinus thrombophlebitis is not preceded by a perisinal abscess or a periphlebitis. The interior of the sinus becomes infected by the extension into it of the thrombi in the smaller venules in the mastoid mucosa and cell walls. Consequently, the outer wall of the sinus will be normal in appearance, since it is not attacked by the disease. This is seen in case 8, wherein at the first operation the sinus wall was inspected and found normal. Yet the continuance of the sepsis spoke for a further involvement of the sinus lumen, and the second operation on the sinus disclosed a thrombus in its interior.

Features in the Laboratory Findings.—The blood culture is a valuable aid in the diagnosis of sepsis. Its limitations, however, must be understood before one can properly estimate its value. In a review of the cases, it will be found that cases 1, 2, 4, 5, 6, 8 and 10 had negative blood cultures. In all but case 10 either a formed thrombus or fluid pus was found in the interior of the lateral sinus. In case 10, no thrombus was found in the lateral sinus. The sepsis here in all probability resulted from a direct infection of the blood stream at the time of the rupture and plugging of the lateral sinus.

Had these cases been permitted to wait until a positive blood culture was obtained, it might have been too late for surgery on the sinus and the internal jugular to have effected a cure. The other signs of sinus thrombosis were sufficiently in evidence for a diagnosis to be established. Nature always attempts to localize an infection. If the thrombus in the lateral sinus is viewed as a protective mechanism on the part of the body to stop the organisms from entering the general circulation, it becomes understandable that the thrombus in cases of coalescent and chronic mastoiditis will be formed as soon as the infection threatens to invade the interior of the sinus. The thrombus may then become secondarily infected; but the infection is situated in the center of the thrombus. The infected portion of the sinus is thus surrounded by noninfected thrombus. Where this infection within the thrombus is successfully localized, none will gain entrance to the general circulation. Should suppuration of the infected portion of the thrombus

ensue, no bacteria will enter the general circulation as long as sterile thrombus surrounds this area. This explains the reason for the negative blood cultures in these cases. Should one procrastinate in operating because of the negative blood culture, however, the entire thrombus may eventually become infected and disintegrated, and particles of the infected thrombus will be released into the general circulation. A positive blood culture would then, of course, be obtained.

Of far more importance than the blood culture is the study of the red and white blood cells and the hemoglobin. In almost every case of sinus phlebitis the total number of white cells and the percentage of polynuclears will be increased. This is true of any infection, with some exceptions, notably tuberculosis and typhoid fever. Where an elevation in the total leucocytosis was noted preoperatively, the white cell count should show a gradual return to normal during the week following operation. The continuance of a high total count and a high polynuclear percentage is indicative of a remaining infection. After all other possible causes for the leucocytosis have been eliminated, a sinus phlebitis should be strongly suspected. Particularly is this true where unilateral fever and headache are present. The same significance should be attached to a leucocyte count which, normal for a time after operation, suddenly begins to rise. Cases 1, 2, 6, 9 and 11 demonstrate the above factors in the reaction of the white blood cells.

In this connection, the von Schilling method of counting the blood cells should be mentioned. While not shown in our cases because of its recent use by us, it affords certain prognostic and possibly diagnostic data. Briefly, the principle is as follows: The polynuclear leucocytes are divided into two groups, those which show a segmented nucleus and those which have an unsegmented nucleus. The former are the adult forms, while the latter are the young polynuclears, in which the nucleus has as yet not undergone complete segmentation. In any active infection the marked increase in the number of polynuclears is also accompanied by the appearance in the blood stream of an increased number of these young forms, called staff cells. While the infection continues active, the percentage of staff cells also continues high. Where the infection shows signs of subsiding, the percentage of staff cells shows a rapid

decrease, regardless of whether the total white count and the polynuclear percentage remain high. Consequently, this method promises an earlier means of judging the progress of an infection. I have had little experience with its value in cases of sinus phlebitis; but in cases of acute mastoiditis the von Schilling count has proved more valuable than the ordinary method of white cell counting. For reference the reader is referred to the excellent papers of Levy² and Heidemann.³

The study of the red blood cells and the percentage of hemoglobin in all cases of otitic infection is likewise important. It assumes its greatest significance in cases wherein a hemolytic organism is the causative agent. The organisms of this type live on the hemoglobin of the blood cells; and, in the event that they have gained entrance to the circulation, a gradual fall in the number of red cells and the hemoglobin percentage is noted. In cases 1, 3, 10 and 11, this gradual drop after the primary operation on the mastoid is well demonstrated. In all the other cases except 8, 9 and 12, a reduction in the number of red cells and the percentage of hemoglobin was noted before operation. These cases were operated upon for sinus thrombosis at the time of the first operation and consequently any further reduction was stopped. In case 8, the hemoglobin and the number of red blood cells did not rise after operation but remained at the level noted before simple mastoidectomy. This, in addition to the septic temperature, indicated the continuance of a septic focus in the lateral sinus. Case 9 was seen after the thrombus had already formed and the sepsis been in evidence. It cannot therefore be used for comparison.

It will also be noted that I use the findings of the red cell and hemoglobin estimation in guiding me in the use of transfusions. Regardless of the clinical picture, the temperature or the blood culture, transfusion is given to the patient only when the hemoglobin is low or shows a gradual reduction in successive counts. For instance, when a patient presents himself for immediate operation on the sinus wherein only one blood count was done and where that count showed a low hemoglobin and a reduction of the red cells, a transfusion is given as soon after operation as possible. On the other hand, a patient who has been under observation for a time and who shows a gradual reduction in the count before operation on the sinus and

the jugular, will receive a transfusion of blood only if the hemoglobin continues to drop after operation. I consider that the only indication for transfusion in otitic sepsis is to replace the hemoglobin that has been destroyed. The transfusion helps the patient in his fight against the invading organism by supplying the patient with fresh hemoglobin.

Correlation of the Various Findings.—In a case that presents an acute mastoiditis, unilateral headache, edema over the mastoid process, reduction in the number of red blood cells and hemoglobin percentage, a sinus phlebitis should be suspected. If, at operation on such a case, an exposure of the lateral sinus is discovered with granulations present on the sinus wall, excision of the infection portion of the sinus wall and obliteration of the sinus will prevent the formation of a subsequent otitic sepsis.

In a case of acute otitis which from the onset shows a septic temperature, gradual reduction in the red cells and hemoglobin count and absence of the classical signs of mastoiditis, a hemorrhagic mastoiditis should be recognized as the cause of the syndrome and an immediate simple mastoidectomy performed. If the operation is done early, it will suffice to thoroughly extirpate all the mass of infected thrombi, thus preventing extension into the lateral sinus.

In a case of chronic mastoiditis due to the dangerous type of lesion causing bone necrosis, the onset of unilateral headache and a profuse discharge from the ear is strongly suspicious of a sinus phlebitis.

After simple or radical surgery on the mastoid process, the occurrence of unilateral headache, increased number of leucocytes, reduction in the red cells and hemoglobin should make one suspect an involvement of the lateral sinus. If these symptoms do not abate, one is justified in operating on the sinus and obliterating it if no other cause is found to account for the symptoms. This holds true, despite the fact that the blood culture may be negative.

Correlation of symptoms in the manner suggested above will lead to an earlier diagnosis of sinus phlebitis and a consequent early surgical intervention. If it is remembered that the obliteration of the lateral sinus in itself entails no danger to the life of the patient and does not add noticeably to the

burden of the patient in his recuperative period, surgeons will be less hesitant to intervene in the face of positive clinical signs and negative blood cultures.

Unusual Features Presented by Cases 2 and 3, in Which the Jugular Was Not Operated Upon.—In case 2, the diagnosis of sinus thrombosis was made on the rise in temperature, the persistent unilateral headache, the high leucocytosis and the reduction in the red cells and hemoglobin. The blood culture was negative. At operation a formed thrombus was found, and, after its removal, free bleeding was obtained from both the distal and the proximal ends of the incised sinus. The operation was completed at this point, and the internal jugular vein was not ligated.

In case 3, the diagnosis of sinus thrombosis was evident prior to operation on the mastoid process, and the blood culture was positive, yielding three colonies of streptococcus hemolyticus to the plate. At operation the sinus was opened and a disintegrated thrombus found. After its removal free bleeding resulted from both ends of the incised sinus. The internal jugular vein was not ligated.

In both these cases the patients made an uneventful recovery. I call attention to the mode of procedure in them not so much for the purpose of bringing into discussion the pros and cons of ligation of the internal jugular vein, but to stress the type of case in which I felt that ligation of the jugular could be safely omitted. One of the cases had a positive and the other a negative blood culture. Both cases were septic clinically. Both cases had a thrombus in the lateral sinus which had not extended to the bulb, and consequently, after the removal of the clot, free bleeding was obtained from the bulbar end. I felt then that I had succeeded in completely exenterating the focus of infection in the blood stream and that therefore there was no need of excluding from the general circulation a focus that no longer existed. The internal jugular vein was not operated upon, and the results justified the procedure. This, however, is not to be recommended as a general rule. Safety lies in the ligation of the jugular; but in selected cases the procedure cited herein may be borne in mind. I do feel that with a more thorough understanding of the value of the blood picture as an indication of the continuance or subsidence of the

general infection, ligation of the internal jugular will not be done in all cases of sinus thrombosis as a routine procedure. Where, after removal of the thrombus and obliteration of the sinus with resultant free bleeding from the bulb, the blood picture shows a return to normal, the jugular operation may be eliminated. Should the blood picture and the clinical symptoms show a continuance of the sepsis, the internal jugular vein can be ligated at a subsequent time.

Unusual Features Presented by Cases 9, 10 and 11, in Which the Sinus Ruptured Spontaneously.—Cases 9, 10 and 11 present an interesting feature. In all three cases no evidence of systemic infection existed prior to the first operation on the mastoid. In cases 9 and 10, a necrosis of the sinus plate was found at the time of the simple mastoid operation. In case 11 the sinus was exposed during radical operation. In the first two cases the sinus ruptured during the removal of the packing from the mastoid wound. In case 11, the sinus ruptured spontaneously after removal of the packing on the operating table. All these cases developed a subsequent sepsis, were operated upon the sinus and internal jugular and recovered. It is difficult to state the reasons for the spontaneous rupture of a sinus which has no thrombus in it and whose wall, upon macroscopic inspection, shows no abnormalities. In case 9 it might be said that the perisinal abscess so weakened the sinus wall that the force of the blood in the sinus broke the thinned and diseased wall, but this could hardly be assumed of the rupture of the sinus in the other two cases, where the wall appeared normal. It must not be forgotten, however, that in case 10 there was found a necrosis of the sinus plate, and this may have produced thinning of the vein wall. It may be that the packing inserted into the wound was too tightly pressed on the sinus wall, interfering with the free flow of blood and causing a local necrosis, or the packing may have become adherent and torn the vein upon being removed. All these are possibilities. In the present state of our knowledge I am unable to assign a specific reason for this happening. It might be well to handle these cases without gauze packing, using only a rubber dam or gutta percha or cigarette drain to lead the pus out of the depths of the wound. I have used this method of draining mastoid wounds for the last two years

with excellent results; but where complications, such as sinus thrombosis, have prevented me from closing the wound I have continued with gauze packing. I shall, however, discontinue the use of gauze packing in the mastoid wound in all cases because of the untoward results in cases 9, 10 and 11.

Unusual Features Presented by Case 12.—Case 12 is an exceptional one. I have purposely omitted including any of the findings in this case from the general discussion. They are so unusual that they warrant special consideration. Following mumps the child developed an otitis with symptoms of meningeal irritation and a septic temperature. The exploration of the mastoid process was undertaken at the request of the attending pediatrician and the neurologist on the case. It was normal throughout. The sepsis continued unabated for twenty-one days. Throughout the entire course a leucopenia was present. The red cells and hemoglobin, however, showed a steady drop, reaching 2,800,000 red cells with 59 per cent hemoglobin.

The child then had two chills and a positive culture of streptococcus hemolyticus was obtained from the blood. A primary thrombosis of the jugular bulb was then diagnosed and a large formed thrombus removed from the bulb.

There are three cardinal signs of primary bulb thrombosis, all of which were present in this case: An acute purulent otitis media, a normal mastoid and a sepsis. Where these signs are augmented by a positive blood culture, the diagnosis is certain. In this case, however, many factors arose to confuse the picture. The persistent leucopenia and the onset with meningeal signs strongly suggested a toxic encephalitis following mumps. Then the persistence of the sterile blood cultures also spoke against the diagnosis of primary bulb thrombosis. The clot, however, was one which was firmly organized and the operation was performed for its removal almost concomitantly with its beginning disintegration. This is the reason for the favorable outcome in this case.

The leucopenia can be explained in one of two ways. The finding of a leucopenia is not uncommon in mumps. A leucopenia is also present in some types of streptococcus infections. One has but to recall the recent epidemic of streptococcus sore throat and the epidemic influenza wherein the streptococcus

played the important rôle. At that time these infections showed a leucopenia. I feel that in this case the type of infecting organism rather than the mumps was the cause of the leucopenia, because the latter persisted after the blood stream was invaded by the streptococcus hemolyticus.

The gradual drop in the number of red blood cells and the percentage of hemoglobin is another outstanding factor in this case. It clearly demonstrated the presence of a hemolytic organism somewhere in the blood stream. Had I relied on this finding as an evidence of a sepsis, I would have explored the lateral sinus and jugular bulb long before the blood culture became positive. I therefore add to the symptomatology of primary bulb thrombosis a gradual reduction in the number of the red blood cells and the hemoglobin percentage.

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- 51 WEST 73RD STREET.

XXVII.

ANGIOMA OF THE AURICLE: REPORT OF CASE.

By JOHN RANDOLPH PAGE, M. D.,

NEW YORK.



Fig. 1.



Fig. 2.

The above photographs are of a girl baby who was seen first at the Manhattan Eye, Ear and Throat Hospital on July 24, 1925, when she was forty days old. Diagnosis: Angioma of the left auricle. The ear at birth was said to have been blue and hard and twice the size of the opposite normal ear. Pulsation in it was noticed by the mother two days after birth. When seen at the hospital the affected auricle was more than twice the size of the normal ear and seemed tense and full of enlarged venous vessels. A large artery entered from below, and the whole mass pulsated. The child was taken to another department and treated with radium from the age of three

to twelve months but without benefit. On its return to my clinic the following July, after a year in the other department, admission to the hospital for observation and operation was advised but refused, and the child was again taken away and not brought back until May, 1927, two years after it was first seen. A small ulceration over the antihelix, which bled profusely at times, seemed to have been the result of the radium. The patch of gauze over this ulceration was not removed at the time the photograph was taken, for fear of bleeding. The ear was then considerably larger than it had been the year before. Consent was finally obtained for operation. Subcutaneous ligation of the vessels at the attachment of the auricle was performed, and pulsation in the auricle ceased for twenty-four hours but later returned, and the auricle became more tense and swollen. As the upper one-half of the helix retained a fairly normal appearance an attempt was made, before resorting to amputation, to construct a small auricle. The enlarged and involved portion of the ear was excised, the upper rim of the helix was drawn around a portion of the concha at the meatus, and a fairly well shaped auricle was fashioned from the part that was free from enlarged vessels. This attempt gave promise of success for the first few days, but a report from the laboratory that the excised portion showed signs of malignancy led us not to delay amputation any further. Because of this ominous report, it was thought advisable to first use bipolar endothermy to coagulate a path of destruction through which could be passed an endotherm knife to remove the auricle. This Dr. Wyeth successfully performed. The coagulation could not be induced or the cutting carried to the full depth of the vessels over the facial nerve for fear of paralyzing the face. A slight recurrence, therefore, has taken place in the lower part over the region where the nerve enters the parotid. It is, however, superficial. Dr. Robinson is of the opinion that one or two treatments with radium will obliterate these vessels. The membranous canal at the time of operation was preserved and sutured at the meatus so that no atresia developed. The tympanic membrane is apparently normal. Hearing also is normal, so far as it can be tested in a child of that age. A later report on the tissue removed was:

"June 16, 1927. Section shows a covering of skin, beneath which are seen numerous blood vessels and a very cellular connective tissue. Diagnosis: Plexiform hemangioma; actively growing.

"June 28, 1927. Dr. James Ewing reports as follows on this case: 'A cellular capillary angioma but not likely to recur if removed surgically. Diagnosis: Angioma.'

"(Signed) Pathologist, A. A. EGGSTON."

127 EAST 62ND ST

XXVIII.

AFFECTIONS OF THE LABYRINTH AND EIGHTH
NERVE IN LEUKEMIA.

By J. S. FRASER, M. B., F. R. C. S. ED.,

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We are indebted to Dan McKenzie of London for his painstaking search of the literature in order to discover the authenticity or otherwise of Ménière's original case. With this acknowledgment it may be permitted to quote from McKenzie's article.

"So stealthily has the story (I had nearly written the 'myth') of Ménière's original case insinuated itself into our otological literature that we are all in the habit of looking upon its data as proved, and have little or no hesitation in basing theories and explanations of various kinds upon them. . . . In the Gazette Médicale de Paris of 1861, p. 598, Ménière published a paper entitled "Observations des Maladies de l'Oreille caractérisées par des Symptômes de Congestion Cérébrale Apoplectiforme," in which he applied to clinical medicine the then recent discoveries of the physiologists on the functions of the semicircular canals. He pointed out that vertigo, which had formerly been regarded as a forerunner or as an actual symptom of apoplexy, was, in reality, produced by disturbances in the semicircular canals induced by disease."

McKenzie has translated Ménière's account as follows: "I have already spoken, *a long time ago* (italics, J. S. F.), of a young girl who, having traveled by night in winter on the outside of a "diligence," when she was at a catamenial period, had, in consequence of a considerable cold, complete and sudden deafness. Her chief symptom was continual vertigo, the slightest effort to move produced vomiting, and death followed on the fifth day. The necropsy showed that the cerebrum, cerebellum and spinal cord were absolutely exempt from any alteration, but as the patient had become suddenly deaf

after having always had perfect hearing, I removed the temporals in order to examine the cause of this complete deafness, so rapidly supervening. The sole lesion I found was the semicircular canals filled with a red plastic material, of which scarcely any traces were perceived in the vestibule, and which did not exist in the cochlea."

In order to discover the *original* description of Ménière's famous case, McKenzie extended his investigation and at last discovered a German treatise on diseases of the ear, by Kramer, which Ménière, in 1848, had translated into French and annotated. On page 397 of this work, the following note by Ménière appears: "I have seen a young girl stricken with complete deafness in the short space of a few hours. Death permitted me to dissect carefully the two temporals and I found in the *whole of the labyrinth* a sort of reddish plastic lymph." McKenzie adds that it is obvious that these two statements concern one and the same case. Yet there is a serious discrepancy in the descriptions. In the *Gazette Médicale* it is stated that the exudate was found only in the canals, whereas in the translation of Kramer's book it affected the whole labyrinth. Moreover, in this, the earlier publication, there is no mention of vertigo. Ménière nowhere calls the lesion in the labyrinth a hemorrhage and yet this is the tradition cherished in our textbooks.

McKenzie concludes that the facts at our disposal are insufficient to enable us to decide what the lesion was in Ménière's famous case, and holds that his opening allusion to it as a "myth" is not entirely unjustifiable.

Whether he quite deserves it or not, Ménière has obtained the credit of being the first to point out that the sudden onset of deafness and giddiness is to be attributed to a lesion of the internal ear or of the eighth nerve. Various theories as to the pathology of Ménière's original (?) case have been adumbrated. G. Alexander of Vienna, for instance, has suggested that Ménière's patient suffered from a bleeding disease, such as leukemia, and that the lesion in the inner ear was produced by a sudden hemorrhage into the labyrinth. Sydney Scott of London, on the other hand, has suggested that the case was one of acute labyrinthitis. The cause of death in Ménière's

case was certainly not proved by the autopsy, as the presence of a hemorrhage or plastic exudate in the labyrinth would not, in an otherwise healthy girl, produce a fatal result. Moreover, there is no reason why a labyrinthine hemorrhage should occur in a normal person. It is, therefore, not surprising that similar cases have never been proved by microscopic examination.

Hemorrhage into the inner ear, even in bleeding diseases, is probably a very rare occurrence. For many years past, the writer has asked his medical colleagues in the Royal Infirmary, Edinburgh, to inform him regarding any case of bleeding disease associated with deafness and/or giddiness, but the four cases described below are the only ones which have been brought to his notice. The writer wishes to acknowledge his indebtedness to Professor Lovell Gulland, to Sir Robert Philip and to Dr. W. T. Ritchie for their kindness.

Case I.—Annie Morrison, aged 40, sent for examination by Professor Gulland, on October 17, 1917. The patient suffered from splenic leukemia and was being treated by arsenic and radiation to the region of the spleen. She had a sudden attack of deafness and giddiness in February, 1917, and could not leave her bed. She stated that at that time the room appeared to go round and that sickness and vomiting were present for six days.

On examination the tympanic membranes were normal. The tuning fork on the vertex was lateralized to the left (good) ear and on the right side Rinne's test was absolutely negative. All the tuning forks were heard by the left ear by air conduction, but none of them were heard by the right ear. The watch was heard at six inches from the left ear but not at all by the right. With the noise box in the left ear the patient was quite deaf. There was no spontaneous nystagmus but the patient swayed on Romberg's test. Rotation to the right produced nystagmus to the left of fifteen seconds' duration, while rotation to the left produced after-nystagmus of only seven seconds' duration. Cold syringing of the right ear produced no nystagmus or giddiness even after two minutes, whereas cold syringing of the left ear produced the usual nystagmus to the right in fifty seconds.

The case was regarded as one of leukemic hemorrhage into the right labyrinth.

Case 2.—David Jeffrey, aged 22 years, was sent for examination by Sir Robert Phillip on the 6th of April, 1918. The case was one of lymphatic leukemia. The patient stated that ten days before his admission he had suffered from a sudden attack of deafness in the right ear, associated with giddiness, sickness and vomiting.

On examination both tympanic membranes were practically normal. On Weber's test the tuning fork was lateralized to the left (better) ear; none of the tuning forks were heard by the right ear. The watch was heard on contact by the left ear but not by the right. With the Bárány noise box in the left ear the patient could hear a shout.

There was no spontaneous nystagmus but well marked swaying on Romberg's test. On walking the patient tended to fall to the right. Cold syringing of the right ear produced no nystagmus or giddiness after two minutes.

This case also was regarded as one of leukemic hemorrhage into the right labyrinth. Neither of these cases came to autopsy.

Case 3.—Alfred G. Page, aged 35, a wire cleaner working with caustic potash, was admitted to Professor Gulland's ward on February 22, 1926. The patient had suffered from weakness and sleepiness for two months before admission. One month before admission he had two teeth extracted and this was followed by profuse bleeding. There had also been slight epistaxis during the past month.

Examination: The skin shows yellow pallor; the speech is slow and the breath foul. Blood count: R. B. C., 1,080,000 per cm.; hemoglobin, 20 per cent; W. B. C., 20,500; polymorphs, 29 per cent; eosinophiles, 5 per cent; myeloblasts, 50 per cent; large lymphocytes, 11 per cent. The spleen is enlarged and there are a few enlarged glands in both groins. There is a slight initial lengthening of the coagulation time of the blood. The teeth are in bad condition. The tongue is dry but clean. The liver is not enlarged. Pulse, 120. Systolic blood pressure, 105; diastolic, 60. The patient is dull, lethargic and has a poor memory. The ocular fundus shows retinitis pigmentosa. The drumheads are normal. Examination by the physician shows that there is definite nerve deafness on the left side but no giddiness. There is slight ecchymosis into the skin over the clavicle on the right side.

Progress: The leucocytosis diminished and on February 24th it was only 18,000. On March 3rd there were definite hemorrhages into retina; W. B. C. still 18,000. Death occurred March 7, 1926. The diagnosis was that of acute lymphatic leukemia.

Postmortem examination showed submucous hemorrhages in the stomach; the aorta showed syphilitic aortitis; the spleen was about six or seven times the normal size; the lymph glands showed no obvious enlargement.

On microscopic examination the bone marrow was packed with myeloid cells. The dura mater showed small whitish nodules the size of a small pea, on the under surface over the vertex. These consisted of masses similar in structure to the bone marrow. The liver, kidneys and spleen also showed infiltration with these cells. The cells are myeloid with a basophil homogeneous cytoplasm and large round vesicular nucleus.

MICROSCOPIC EXAMINATION OF LEFT EAR.

Middle ear: The middle ear spaces show a slight amount of serous exudate, especially in the niche of the oval window. The submucosa also shows some small cell infiltration. Inner ear: There is no hemorrhage or small cell infiltration in the endo- or peri-lymphatic spaces. Corti's organ is fairly well formed but the cells of the spiral ganglion appear rather atrophic. The vestibular ganglion in the internal meatus shows some small celled infiltration. The semicircular canals are normal and the cristæ are well formed.

Right ear: On this side also there appears to be well marked atrophy of the cochlear ganglion cells. Corti's organ is not well formed.

Case 4.—Walter Brown, male, aged 25, a dairyman, was admitted to Dr. W. T. Ritchie's ward on February 13, 1926, complaining of weakness of three weeks' duration. He had suffered from dimness of vision for ten minutes on January 31, 1926, from pain in the left shoulder on February 26, 1925, from shivering followed by a skin eruption February 9, 1926. On February 11, 1926, he complained of abdominal pain, and on the day of admission he cut himself while shaving and bled for a long time.

Examination on admission showed bleeding from gums and tongue and subconjunctival hemorrhage in the right eye; temperature 99; pulse 76; lymphatic glands all enlarged and also the spleen; liver enlarged. Blood count showed R. B. C. 5,300,000 per cm.; hemoglobin 80 per cent; W. B. C., 132,500; large lymphocytes 92 per cent. Urine dark brown in color and contains blood; feces dark in color and show blood on testing. Blood cultures revealed no growth (February 15, 1926).

Subsequent history: Bleeding continued from the kidneys and gums. The patient was transfused with citrated blood on two occasions and improved slightly. On February 28, 1926, dimness of vision and some deafness were noted by the physician. Ophthalmoscopy revealed a large retinal hemorrhage in the left eye. The face and eyelids became puffy on March 1, 1926. Death (March 2, 1926) was due to pneumonia.

Note.—An interesting feature of the case was that the white cell count, which was very large on admission, rapidly lessened, and on the day before death was only 1,800 per cm.

Postmortem examination showed numerous hemorrhages into the pericardium, esophagus, stomach; the spleen was greatly enlarged and weighed 720 grams; there was markedly lymphatic hyperplasia, including the tonsils, cervical, axillary and lingual glands; the bone marrow showed extensive proliferation of lymphoid cells on microscopic examination.

MICROSCOPIC EXAMINATION OF LEFT EAR.

Middle ear: The tympanic membrane and ossicles are normal. The submucous tissue of the bony portion of the eustachian tube is greatly infiltrated with small cells. The mucosa lining the antrum and mastoid cells is somewhat thickened and is also infiltrated with small cells. Inner ear: The marrow spaces in the bone around the labyrinth are very cellular. There is no hemorrhage or leucocytic infiltration to be seen in the endo- or peri-lymph spaces of the cochlea, vestibule or canals. Corti's organ is not well formed and the pillar cells can be made out with difficulty, especially in the upper coil. The cochlear ganglion cells appear shrunken. The neuro-epithelium of the utricle is not well formed, but the sacculus shows a more normal condition. The cells of the vestibular ganglion in the internal meatus show less change than those of the cochlear ganglion. The facial, cochlear and vestibular

nerves in the internal meatus appear quite normal. The ductus endolymphaticus is healthy.

Microscopic examination of the right ear shows practically the same conditions as on the left side. There is apparently well marked atrophy of Corti's organ and of the cochlear ganglion cells.

Remarks: It will thus be seen that in these two latter cases (Nos. 3 and 4) of leukemia, both of which suffered from deafness, there was no evidence of hemorrhage into the hollow spaces of the labyrinth. It is true that, in contrast to the two earlier cases (Nos. 1 and 2), which did not come to autopsy, there was no history in these latter patients of the apoplectic onset of Ménière's syndrome, such as one would have expected in a case of hemorrhage into the inner ear. The writer, however, after waiting for many years, in the hope of obtaining the temporal bones from a case of leukemic deafness, thought that it would be worth while to carry out a microscopic examination of the middle and inner ears in these two cases, in order to ascertain the cause of the deafness noted by the physician. Atrophic degeneration of the ganglion cells and nerve endings were the only findings worthy of note.

CASES OF LEUKEMIC DEAFNESS AND GIDDINESS RECORDED
IN THE LITERATURE.

Politzer, in 1884, reported a case which showed connective tissue and bone formation in the perilymph space of the cochlea, vestibule and canals. In addition there were collections of lymphocytes in the spiral ligament of the cochlea and also in the vestibule and canals. G. Alexander, who has observed a large number of cases of leukemic deafness, has in some of them found new connective tissue and bone formation and deposition of pigment, in addition to hemorrhages into the tympanic cavity and inner ear. He has also noted degenerative changes in Corti's organ, in the cells of the spiral ganglion and in the eighth nerve itself. Aage Kock has examined a case of leukemic bleeding into the inner ear in a man of 32 years, who had suffered from malaria. The case was diagnosed as one of splenomedullary leukemia, and the patient had had a sudden attack of giddiness, deafness and noises in the ear. Microscopic examination of the left ear showed recent hemorrhage

into the cochlea, especially in the basal coil. The hemorrhage was found especially in the scala vestibuli of the cochlea, in the vestibule and in the endo- and peri-lymphatic spaces of the canals. There was also hemorrhage into the tympanic cavity. The right ear showed even more marked changes, both the scala vestibuli and the scala tympani being completely filled with blood and the basilar membrane for the most part broken through. Aage Kock notes that the marrow spaces of the ossicles were filled with lymphoid cells. Kepruska's first case was that of a patient who had an acute onset of Ménière's syndrome. Microscopic examination showed small celled infiltration and new connective tissue formation between the fibers of the eighth nerve. In Kepruska's second case the mucous membrane of the tympanum was infiltrated with leukocytes and thickened. In the membranous labyrinth there was only slight infiltration of small cells, but in the internal meatus there was a more marked infiltration, especially in the area where the cochlear nerve enters the bony canals. Tadokoro's patient was a male, aged 47 years, who had had a Ménière attack forty days before death. He was totally deaf in the right ear. Microscopic examination showed leukemic infiltration of the mucous membrane of the middle ear, hemorrhage in the tympanum, in the semicircular canals and in the middle coil of the cochlea, with destruction of Corti's organ. Mishima, in a patient of 23 years, suffering from leukemia, found hemorrhages in the scala tympani of the cochlea and also in the vestibule and canals. There was atrophy of the eighth nerve, with leukemic infiltration between the fibers. Lindt has examined the temporal bones of a male, aged 35, who had a sudden Ménière attack with absolute deafness. Blood examination had proved the presence of leukemia and, at the autopsy, an enormous tumor of the spleen was found. Microscopic examination showed complete destruction of the whole membranous labyrinth. Rudberg has described the case of a male, aged 44, suffering from myelogenous leukemia. On microscopic examination it was found that one ear showed acute leukemic infiltration of the labyrinth and eighth nerve; in the other ear the changes were of a more chronic nature.

Finlayson records the case of a female, aged 29, suffering from splenic leukemia. A blood count showed 2,000,000 R. B.

C. per cm. and 1,190,000 leucocytes. One month before admission to hospital she suffered from deafness, tinnitus, giddiness and vomiting. Clinical examination showed hemorrhage beneath the conjunctiva and also into the fundus oculi. On examination during life the middle ear appeared to be normal. Nerve deafness was present. Postmortem macroscopic examination of the labyrinth showed hemorrhages into the vestibule and first turn of the cochlea. Gimplinger, in a case of chronic leukemia, found accumulations of small cells in the hollow spaces of the labyrinth and organized connective tissue and new bone formation, especially in the scala tympani of the cochlea. Nishio examined the temporal bones of a female patient suffering from myeloid leukemia. The middle ears were normal. The hollow spaces of the labyrinth showed otitis interna ossificans, with the formation of new connective tissue, osteoid substance and, finally, new bone. Nishio thinks it possible that hemorrhages first occurred in the inner ear and that the new formation of connective tissue and bone resulted.

Leukemic hemorrhage into the labyrinth is, of course, by no means the only cause of Ménière's syndrome; indeed, it is a very rare condition. During twenty-one years only two cases diagnosed as leukemic hemorrhage into the inner ear (Cases 1 and 2, *vide supra*) have been seen at the Ear and Throat Department of the Royal Infirmary, Edinburgh, formerly under the charge of Dr. A. Logan Turner, and now of the writer. During this period about 94,200 patients were examined at this department; of these, 33,500 were suffering from an affection of the ears.

Apart from lesions of the inner ear, such as diffuse labyrinthitis, the onset of giddiness and deafness may be due to a toxic neuritis resulting from septic absorption from the alimentary tract or from other foci of infection, such as the teeth; indeed, this condition appears to be by far the most common cause of Ménière's syndrome. Herpes zoster oticus, neuritis following exposure to cold, syphilitic affections of the ear, mumps, cerebral arteriosclerosis, chronic nephritis and tumors of the cerebellopontine angle may all produce giddiness and deafness. A sudden increase in intralabyrinthine pressure—the so-called "glaucoma of the labyrinth" (Jenkins, Portmann and others)—or accumulation of cerebrospinal fluid

in the lateral cistern (Bárány) may also produce Ménière's syndrome. It is rare in cases of otosclerosis to obtain a history of giddiness, though deafness and noises in the ear are, of course, the most prominent symptoms.

Certain otologists have drawn an analogy between Ménière's disease and cases of asthma and epilepsy. The suggestion is that patients with one or other of these troubles suffer from faulty internal chemistry and that possibly a sudden flooding of the blood with a noxious substance may produce in one person an attack of asthma, in another an epileptic seizure and in a third Ménière's syndrome. These conditions have, of course, been regarded as "nerve storms." Such suggestions are still very much "in the air," and it is probable that we have still a long time to wait before our knowledge of biochemistry increases to such an extent that we can do more than theorize upon this subject.

Cases presenting Ménière's syndrome may be divided into three classes: (1) Those which have an apoplectiform onset and result in total deafness and loss of vestibular function—e. g., leukemic hemorrhage, diffuse purulent labyrinthitis and some cases due to mumps and congenital syphilitic infection. (2) Those with sudden onset, *which is not by any means always* followed by complete deafness or loss of vestibular response—e. g., toxic neuritis, herpes zoster oticus, "glaucoma of the labyrinth" and Bárány's syndrome. (3) Those with a more or less gradual onset—e. g., cerebral arteriosclerosis, tumors of the eighth nerve and some cases of otosclerosis and of the late form of congenital syphilitic deafness.

CONCLUSION.

In bleeding diseases, especially in leukemia, it occasionally happens that hemorrhage occurs into the hollow spaces of the labyrinth, with the sudden onset of the well known Ménière's syndrome. Subsequent clinical examination reveals complete deafness and loss of vestibular response in the affected ear. If death occurs soon after the attack microscopic examination shows hemorrhage into the labyrinth. If the patient survives for a time after the Ménière attack and dies later, microscopic examination of the inner ear shows the condition known as "otitis interna ossificans," in which there is new formation of

connective tissue and bone in the hollow spaces of the labyrinth, with atrophy of the eighth nerve and of the nerve endings. Microscopic examination of the brain has, in some cases at any rate, shown degeneration of the nuclei and nerve fibers along the intracranial course of the eighth nerve. Apart from hemorrhages into the labyrinth, deafness in cases of leukemia may be due to degenerative changes in the spiral ganglion cells and nerve endings.

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XXIX.

THE MONOCHORD IN UPPER LIMIT DETERMINATIONS AS AN ADJUNCT TO AUDIOMETRY.

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A loss in acuity for tones near the upper limit of audibility is classically interpreted as being indicative of a lesion of the auditory nerve or of the perceptive apparatus of hearing. The microscopic examination of ears which in life have presented this type of loss should reveal certain characteristic defects. Moreover, if a resonance theory of hearing is to be assumed, these defects should be found in the basal coil of the cochlea or in certain portions of the auditory nerve. Analysis of the tests used in making these determinations makes it seem quite probable that what may be considered an actual deafness for these high tones is but a loss in sensitivity.

The determination of the acuity of hearing for these tones implies, first, that the tones shall be produced for observation; and second, that intensity measurements should be made in order to determine the threshold of sensitivity. They should permit the same qualitative and quantitative determinations as do those in the lower portion of the tonal range. Recent advances in audiometry have made possible the measurement of the lower range of tones, but if the upper limit of audibility be in the neighborhood of 20,000 d. v., no audiometer presented offers the possibility of a survey of the tones near the upper limit. For example, the studies which have from time to time been presented by the writer have been limited to the range of tones below 7,070 d. v., while the 1A audiometer of the Western Electric Company produces twenty-two octave and semi-octave tones between 32 d. v. and 16,384 d. v.

The available instruments which are capable of producing these high tones for observation are the Galton whistle, the Koenig bars, and the monochord modified by Struycken, but

none of these offers the possibility of making an intensity measurement unless that of distance be used. This is impracticable, first, because conditions of testing usually necessitate a limitation of space; and second, the elimination of reflection, standing wave effects, etc., would require prohibitive construction. The monochord does offer the possibility of making comparisons of the upper limit of audibility by air conduction and by bone conduction, which add definite information concerning the functioning power of the receptors for these high tones.

The following records are selected from a large group as being illustrative of the facts which may be determined by the use of the monochord in this manner.

Figure 1 is the record given by a woman, 45 years of age, in the examination with the 1A audiometer. Her clinical diagnosis was otosclerosis. She gave a history of deafness, which had been gradually progressive for more than twenty years. The audiogram* shows that the highest tone audible in each ear was 5,760 d. v. It was ascertained with the monochord that she could hear to 11,000 d. v. in each ear by air conduction, and to 21,000 d. v. in the right and to 23,000 d. v. in the left by bone conduction.†

The rarity with which observers in late adolescence or early adult age hear tones higher than 19,000 d. v. or 20,000 d. v., with the monochord as a sound source, has led to the belief that this is about the normal upper limit for this instrument. If this be true, it is not probable that any defect in the perceptive apparatus could have been found in this case if histologic sections could have been made.

The high tones produced by the 1A audiometer, even at maximum intensities, cannot be considered loud. The higher upper limit secured with the monochord by air conduction in this case may be attributed to the fact that the tones from the monochord for these pitches are of greater intensity than the same tones from the audiometer. The further elevation of the upper limit by bone conduction makes it appear probable that

*The copyrighted charts are used with the permission of the Bell Telephone Company Laboratories.

†In the records presented, the observations for the right ear are indicated by circles and those for the left with crosses. Arrows indicate tones not heard at maximum intensities.

if tones of sufficient intensity could be produced for air conduction a normal upper limit would have been obtained in this case.

Figure 2 is the record given by a woman, 38 years of age, who in addition to a cardiac condition had a 4 plus blood Wassermann. She gave a history of having had earache without discharge in the left (better hearing) ear two years prior to the time these tests were made. It will be noted in the audiogram that she hears to 13,004 d. v. in the left ear but did not hear 16,384 d. v., the highest tone produced by the A1 audiometer, at its maximum intensity. Both high and low tones were inaudible in the right ear, 4,096 d. v. being the upper limit of audibility. The test with the monochord showed that she could hear 12,000 d. v. in the left ear by air conduction and 15,000 d. v. by bone conduction. With the left ear masked, she was able to hear the longitudinal vibrations from the monochord when set at the lowest pitch (5,500 d. v.) by air conduction, and by bone conduction to 6,000 d. v. This close correspondence of results from the two instruments makes it seem probable that a microscopic analysis would reveal a considerable defect in the perceptors for these high tones, especially in the right ear. On the other hand, if it were possible to produce tones of still greater intensity than those produced with the monochord, it cannot be denied that this upper limit might be still further raised. Until such instruments have been produced and studies made, conclusions must necessarily be drawn from the available data.

Figure 3 is the record given by a woman, 65 years of age, with a diagnosis of hypertension and syphilis. She denied all specific ear trouble, but stated that she had a little difficulty in understanding conversation when she used the telephone. The 1A audiometer shows the upper limit to be 5,760 d. v. in each ear, while with the right ear she could hear the monochord to 13,000 d. v. by air conduction and to 15,000 d. v. by bone conduction, and with the left ear she could hear to 13,000 d. v. by both air conduction and bone conduction.

Assuming from such records as that shown in Figure 2 that the frequency calibration of the monochord is accurate, the disparity in the upper limits shown by the two instruments in the case given in Figure 3, can be accounted for only by the fact that the tones from the monochord are much more intense than those produced by the audiometer.

Records showing such losses are frequently found in examining the hearing of persons in advanced age, those with generalized vascular lesions and those with syphilis. It may also appear in records from persons of earlier adult age. Figure 4 is the record given by a man, 45 years of age, who had a diagnosis of spinal cord tumor. He gave a history of bilateral otitis media occurring in early childhood and intermittently, as he became older, the last attack being when he was 31 years of age, at which time the right ear discharged for about one week. He considered his hearing perfectly normal at the present time. The audiogram shows that the hearing was somewhat poorer in the right ear than in the left, but that 5,760 d. v. was the upper limit for each ear. The upper limit shown by the monochord was 5,500 d. v. for the right ear and 6,000 d. v. for the left ear by air conduction, while the upper limit by bone conduction was 6,000 d. v. in each ear.

Another case illustrating this marked lowering of the upper limit in adult life is shown in Figure 5, an audiogram made with the 2A audiometer, which gives but eight tones. The patient was a woman, 21 years of age, with a diagnosis of pulmonary tuberculosis. She gave a history of earache without discharge in childhood and stated that she sometimes experienced difficulty in understanding conversation. The audiogram shows that 4,096 d. v. is the highest tone audible in each ear. The monochord showed the upper limit to be 5,500 d. v. in the right ear and 6,000 d. v. in the left. The upper limits by bone conduction were the same as those by air conduction.

Conclusions: The upper limits of audibility ascertained by use of the monochord add rather conclusive evidence as to the functioning power of the perceptive and conductive mechanisms of the ear. This is possible because the upper limit tones are produced with greater intensity than similar tones produced by the audiometers which are available to otologists, and because it offers the possibility of securing determinations by means of bone conduction. Certain cases showing a marked lowering of the upper limit by air conduction can be shown to have a normal upper limit by bone conduction, which certainly eliminates any thought of a destruction of the nervous mechanism for the perception of these tones.

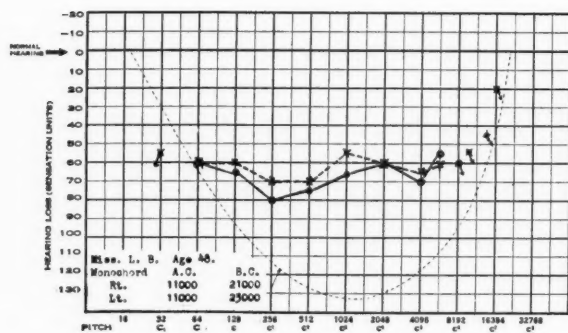


Fig. 1. Otosclerosis.

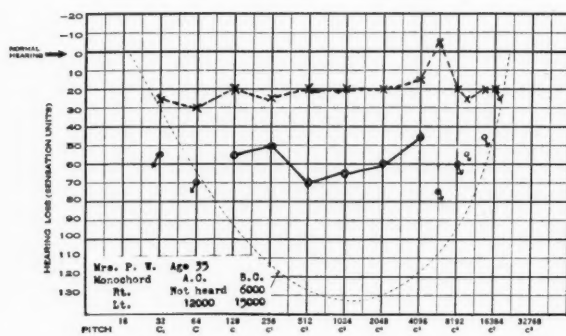


Fig. 2. Cardiac. 4+ Wassermann.

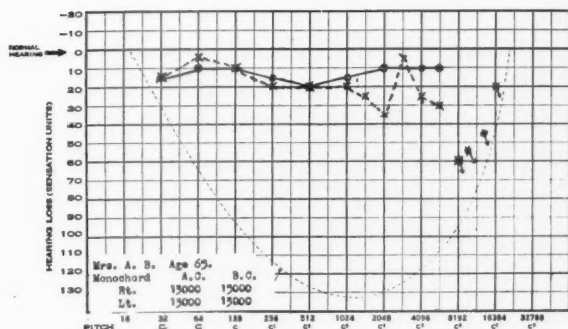


Fig. 3. Hypertension. Syphilis.

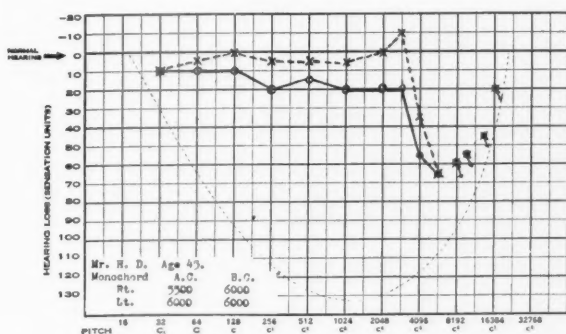


Fig. 4. Spinal cord tumor.

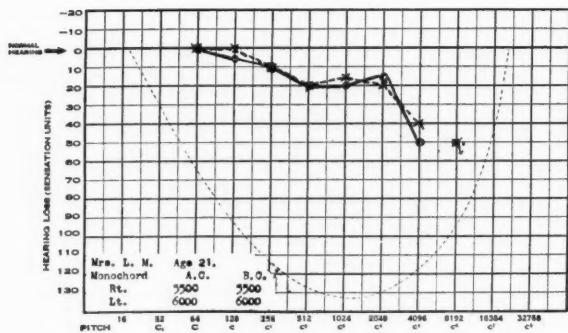


Fig. 5. Tuberculosis.

XXX.

EVOLUTION OF THE ACOUSTIC METHOD.

BY MAX A. GOLDSTEIN, M. D.,

ST. LOUIS.

There has been so much misunderstanding of what we have termed "The Acoustic Method" as a definite means for the conservation of residual hearing that a clear and comprehensive presentation may be a timely consideration.

The idea of stimulating the auditory nerve of the congenitally deaf child by means of sound vibration is a very ancient one. Archigenes, in the first century, advocated the use of the hearing trumpet to intensify sound in cases of deafness. Alexander of Tralles, in the sixth century, and Guido Guida, in the sixteenth century, suggested the stimulation of latent hearing functions by the use of various noises and loud shouting in the ear.

Ernaud, in 1761, demonstrated before the Academy of Sciences, in Paris, that a selected group of the deaf with residual hearing and a comprehension for vowels and consonants could be taught to differentiate these individual sound elements. He was able to improve the hearing of such pupils to include a comprehension for words and, in sporadic instances, he even obtained hearing for sentences.

Ernaud claimed that total deafness in congenital cases did not exist, and this contention, with some modification, we are able to substantiate by observation and tests in large groups of congenitally deaf pupils today.

In 1767, Periere announced that nearly all deaf subjects could be trained to hear words, providing some remnant of hearing was demonstrable at the outset. Periere used a peculiarly constructed hearing trumpet.

Itard, in France, and Toynbee and Wilde, in England, were among the pioneers in otology to recognize the importance of this form of special stimulation for the development of residual hearing.

Urbantschitsch, in the early nineties, planned a system of acoustic exercises by which such stimulation was offered not only to the so-called semi-deaf but also in selected cases of congenitally totally deaf children.

My personal interest and support in the efforts of Urbantschitsch, of Vienna, and my participation in the daily practice of the Döbling Institute, while these exercises were being tested out and carefully studied, was rewarded by the request of Urbantschitsch that I introduce his work in America.

More than three decades have passed since my first observations of the results of systematically applied acoustic exercises have been made. The problem has been seriously studied, critically tested and carefully applied, and the fundamental principle of vibratory stimulation, on which the success of this work depends, has been generally recognized as having a practical and definite place in the pedagogy of the deaf. The experiments introduced by earlier investigators have been much modified, modernized and elaborated, and an effort has been made, in establishing a system of progressive exercises, to comply with the demands of speech training and a better understanding of the physiology of the ear and the psychology of the deaf child.

The name "Acoustic Method" has been selected to dignify and distinguish this special pedagogy from the "Oral Method," the "Sign Method" and the "Manual Method."

At the International Conference on the Education of the Deaf Child, in London, July, 1925, I proposed the following definition of the acoustic method: *Stimulation or education of the hearing mechanism and its associated sense organs by sound vibration as applied either by voice or any sonorous instrument.*

This definition in its comprehensive form includes:

- a. Voice and musical sounds directed through the physiologic tract of the ear to the peripheral or central auditory areas.
- b. Sound vibration as sensed by tactile impression to interpret pitch, rhythm, accent, volume.
- c. Analysis of speech sounds by tactile differentiation.
- d. Synthesis and speech construction by tactile impression.
- e. Sound waves and their significance as appreciated by optical perception.

An additional value of the inclusive expression "*Acoustic Method*" is to clear the atmosphere of a confusion of terms as "auricular training," "aural gymnastics," "aural method," and to embody all recent research, such as radio and telephone principles, inter-relation of the special-sense organs, and a wider appreciation of the physiology of hearing.

It has been frequently observed and herein substantiated that at least 70 per cent of children with biologic congenital deafness retain some remnants of hearing. In this survey there must be excluded the cases of pathologic congenital deafness due to syphilis, rickets, tuberculosis, intrauterine hemorrhage, etc. This remnant of hearing is the objective of this special pedagogy, and it is in the cases of biologic congenital deafness that we may expect our most promising results.

Earlier in our work we used the loud human voice exclusively, but found that this excessive volume of tone was objectionable to the sensitive ear of the deaf pupil and ruinous to the voice of the instructor.

As the human voice, especially in its conversational quality, was found to be the main utilitarian value in our exercises with this class of cases, a working system was evolved, the various steps of which form the basis of these training exercises.

For a comprehensive survey we enumerate these steps as follows:

- (1) Vowel differentiation—*a* as in father, *o* as in old, *e* as in eel and *u* as in ale;
- (2) Introduction of the first consonant elements *sh* and *s* and their combinations with the vowel elements;
- (3) Diphthongs are added to the hearing vocabulary of the pupil. The consonant palatal elements *m* and *n* are next applied in their combinations with initial and end-vowel elements;
- (4) Intensive and frequent drills of words made up of these elementary sounds of vowels and consonants are produced in their varying combinations;
- (5) The remaining consonant elements are added in specially arranged successive exercises and in relation to their range of auditory perception. Finally, words, phrases and sen-

tences are built of speech element combinations in the order in which they have been practiced.

It is quite evident that theoretically and practically acoustic exercises heretofore have been directed almost exclusively to the semi-deaf; the pupils regarded as totally deaf have rarely been given these opportunities. Of all authorities who have investigated and developed this work, Urbantschitsch has been perhaps the first who has persisted in his training of the profoundly or totally deaf child by the acoustic method. I find no literature to permit the conclusion that American instructors of the deaf or investigators into this problem have ever before given a fair trial to the totally deaf child by acoustic training.

In planning our course of training by the acoustic method in Central Institute for the Deaf, we have directed that every pupil in the Oral School shall receive daily systematic training of this character, irrespective of the degree of deafness, the age of the pupil or scholastic status. We have devised specially arranged, individual record charts and here every step in the progress of the pupil is noted. We believe that a careful analysis of these charts will gradually lead us to develop a comprehensive working system which will enable all instructors to be placed on the same pedagogic plane and all deaf pupils to be given equal opportunity for development by such training.

Let me emphasize again that it is my sincere opinion that the principal reason for the many unsuccessful attempts and indifferent results in the use of the acoustic method are due to the desultory, aimless and unsystematic form of procedure which has discouraged teacher and pupil alike and which has given rise to so many misconceptions and misunderstandings about this particular special pedagogy.

The construction of a graduated, systematic series of exercises and tables, based on the underlying principles of sound-perception by the normal ear, and studied in conjunction with the pathology and psychology of the deaf child, is here offered as a working plan. At the outset, I admit its imperfections, for it is a difficult task to plan a pedagogic system where a vast array of conflicting problems and theories must be faced; where much of the physiology and pathology of the acoustic

mechanism is still but vaguely understood; where a comparatively short time for practical study and impartial analysis has elapsed and where large groups of pupils by many teachers with varying degrees of pedagogic skill and different pedagogic ideas have not yet been called into consultation to summarize the end results and establish its practical potentiality.

To simplify our work and concentrate our observations we formerly employed two musical instruments at Central Institute—the harmonium for passive education and the human voice for active education.

The harmonium,* or modified accordion,† had a range of six octaves of whole tones from contra C to C-4. For this instrument we chose the blower type of reed, since the tones produced by such a reed may be intensified to a greater degree than with the suction type of reed. Since the introduction of the radio we have practically abandoned the harmonium.

All pupils, regardless of the residuum of hearing, should be given at least ten minutes' daily instrumental stimulation in addition to the acoustic exercises for developing appreciation of spoken language. This instrumental practice should be continued throughout the entire course of acoustic training, for by its use the entire range of the organ of Corti is constantly subjected to stimulation, and inactive or dormant tone islands awakened to greater potentiality. We concentrate on the development of tone-perception within the range of the speaking or singing voice, but in some cases where the pupil shows no perception for tones within this range, we have been able to stimulate such perception by constant application of sonorous vibrations on tone-islands of hearing outside of this range and gradually developing or educating a perception for tones within the range of the speaking voice.

When tests are first made, the classification of the child's degree of deafness will almost always fall into one of the following groups:

Group 1. NO SOUND IMPRESSION WHATEVER FOR ANY NOTE IN THE ENTIRE RANGE.

In this class we would expect to find pupils who give a history of distinct congenital deafness, perhaps a record of heredity or consanguinity in marriage; often two or more deaf children in the family. This first test with the harmonium would also be logically conclusive where negative reaction to tests of the static labyrinth was found and where all functional tests of hearing indicated no sound perception. Profound or total deafness.

*Devised by Victor Urbantschitsch.

†Devised by Max A. Goldstein.

Group 2. AN INDIFFERENT OR CONFUSED SOUND IMPRESSION FOR ONE OR MORE NOTES IN SOME PART OF THE SCALE.

Somewhere in the range the pupil responds to one, two or three notes. These notes are frequently within the limitations of C to C¹. They are heard indifferently or indistinctly and no ability to differentiate these sounds is apparent. This group may be occasionally traced to that class of pupils who have sustained some serious constitutional, febrile, toxic attack, in early infancy, such as meningitis, influenza, convulsions, etc. We might assume that such a meagre perception of sound is associated with a degeneration of the auditory nerve, either centrally or peripherally. Somewhere in this tract a few nerve fibers and cells may have escaped the ravages of such toxemia or degeneration and may still respond weakly to an intense sound stimulation.

Group 3. A FAIR SOUND IMPRESSION WITH SOME ABILITY TO DIFFERENTIATE PITCH OF A SMALL NUMBER OF TONES.

We are now approaching the otologic classification made by Bezold of tone islands. These tone islands are found in the defective labyrinth varying in degree and in position. Bezold subdivides tone islands into five groups when testing deafmutes. Group 1. A tone island in which the hearing range does not exceed two and one-half octaves. Group 2. Where there are several tone perceptions at intervals in the musical scale. Group 3. A loss of hearing for the entire upper scale limit and fair hearing for the lower scale. Group 4. Defects of varying degree in the appreciation of sound impressions of tone vibration in the lower scale.

When the perception for this small group of sounds does not extend over a range of one octave, we are still confronted with a profound type of deafness. We have, however, a tangible starting point for our further acoustic practice. The differentiation of pitch of the tones perceived by the pupil may by repeated practice be greatly accentuated and the perception of tone range surprisingly extended. Here we often find the cases of pupils who have been originally endowed with normal hearing and who, at an early age, have become deaf through profound attacks of scarlet fever, measles, smallpox, diphtheria, etc. The fact that such pupils show a tendency to respond to graduated acoustic stimulation would seem to prove that the toxic degeneration or pathologic changes that have been sustained by the auditory nerve apparatus, either centrally or peripherally, may still be subject to reconstruction, reeducation or regeneration.

Group 4. FAIR SOUND IMPRESSION AND FAIR ABILITY TO DIFFERENTIATE TONES IN SEVERAL SECTIONS OF THE WHOLE RANGE.

Here we find multiple tone islands or multiple areas in the cochlear scale that still respond to fairly normal sound impressions. Pathologically considered, the destructive or degenerative changes which have taken place in such an auditory end-organ must have been of an

irregularly localized character whereby some of the nerve structures of the cochlear scale have been left unscathed at intervals and the rest invaded. For, in using the notes of the harmonium in such a case, it will be found that the pupil responds to a group of sounds of a tone island in the bass cleff, fails to perceive the tones of an octave or more in the middle register and then perceives a group of tones in the C^1 or C^2 octave. In this group progress by both active and passive impressions of the applied acoustic method is often surprisingly rapid.

Another observation which I desire make at this point is: Where tone islands can be definitely demonstrated by tests with the harmonium or the continuous tuning fork series, we are justified in the conclusion that the pathology in such cases is in the peripheral acoustic apparatus and not in the auditory nerve centrum, for if the theory of Helmholtz remains unshaken and if the organ of Corti is the selective peripheral acoustic apparatus by which individual musical tones are analyzed, the perception of tone islands and the gaps of deafness in such a given case indicate that some of the arches of Corti with their associated ganglionic cells and hairs of Hensen still function normally, and other groups of such cells and hairs have been destroyed. In such cases, however, it would seem that the auditory nerve centrum must be active in function.

Group 5. PARTIAL PITCH PERCEPTION FOR NOTES IN SEVERAL AREAS OF THE RANGE.

In this class we would include the group heretofore designated as the semi-deaf. Many of these cases may not even prove to have an auditory nerve pathology, but may have sustained some serious impairment to the sound conducting apparatus—destruction of the drum membrane or ossicles, operative inroads following mastoid or tympanic cavity disease, etc.

ACTIVE ACOUSTIC EDUCATION.

Active Acoustic Education is divided into two processes: Analytic and Synthetic Acoustic Exercises. Through Analytic Exercises we develop the ability to interpret vowels, consonants and syllables as actual auditory impressions independent of the association of ideas and word imagery. The auditory comprehension of language is developed through Synthetic Acoustic Exercises.

OBSERVATIONS AND DEDUCTIONS.

Persistency in Application of Acoustic Exercises.

We have frequently observed the perception of a harmonium tone, of a sustained vocal sound or of a word only after daily and painstaking repetition of the same exercise, continued for weeks and sometimes months. However, when such sound impression is once heard by the pupil, successive exercises progress with less difficulty. Even when the stage of word hearing has been reached, it may require numerous

repetitions before the pupil makes accurate response and before the word image is definitely impressed.

Awakening of the First Sound Perception.

The awakening of the pupil to the first sound impression is usually sluggish, be the sound that of a musical instrument or of the voice. The sound is an unfamiliar thing, the physiologic stimulation is a weak one, the mental impression has not yet been registered. We experience the same reaction in lesser degree in normal hearing. A sound which is unfamiliar to our ear, of slight volume and unknown mental registration, must be repeated to a normal ear before it acquires an accurate interpretation.

Sound Comparisons.

Many of our educational impressions by all of the senses are the result of comparisons. We are taught color, form, size, by comparison; we appreciate heat or cold, rough or smooth, light or heavy, by comparison. We hear loud or soft, high or low, by comparison. So, too, we develop the acoustic method by comparison. We have elicited our first sound impression, whether by harmonium or voice. It may be the harmonium tone C, or it may be the sustained vowel A. To the pupil this is frequently but a sound impression without definite character. We now introduce our comparative element; let it be the harmonium tone G or the sustained vowel O. Sometimes this second step will convey an immediate comparative impression to the pupil. Or, the pupil will be unable to distinguish between the second sound and the first. To him it may be simply a sound impression without intensity, without pitch, without timbre, without direction. The dormant auditory mechanism must of necessity be slow of stimulation or of development. Many of the delicate histologic structures in the organ of Corti may have become so inert from disuse that little or no impression can be made, and such minute stimulation must be repeated again and again before a sufficiently tangible stimulation and reeducation has been established to yield a practical auditory result. Comparative sounds conveyed to the ear seem a logical form of stimulation, for we thereby educate both passive and active impressions by the same effort. The repetition of the same musical sound wave produces a mechanical and physiologic stimulation in the cochlear end organ; comparative musical sounds awaken a mental differentiation in the brain as well as a physiologic selective differentiation in the organ of Corti.

Word-hearing and Word-imagery.

It is one thing to establish word-hearing; it is quite another thing to develop word-imagery. One is physiologic; the other psychologic. Word-hearing depends on the accuracy with which the selective end organ in the cochlea responds to sound combinations; word-imagery is developed only when word-hearing has been associated mentally with the object or thing to which such word applies.

It is good practice, as shown in the exercise tables, to group a series of words of similar sounds: bowl, pole, mole, coal. Association of ideas is as important in this practice as it is in the development of lip-reading. To the hard of hearing pupil a lip-reading exercise containing words of similar combinations and of similar spoken form is understood more accurately when the meaning of the word is fitted to the phrase in which it occurs. A word spoken independently is more difficult to comprehend even by a normal ear than a word that is part of a phrase.

Another point not to be overlooked is the scholarship of the pupil. It is difficult for a pupil with defective hearing to comprehend a spoken word, the meaning of which is unknown. Normally, it is even difficult for the healthy ear to hear words in quick succession where the sentence is meaningless. For instance, if a normal-hearing person is addressed in a foreign language, the words are unintelligible, not because they fail to register in the ear, but because they fail of comprehension in the brain.

As the faculty of association of ideas develops by practice, the hearing takes on more rapid response. Word-hearing and word-imagery succeed each other automatically in normal cerebration, and in our acoustic practice we strive for such normal association of word and idea. The more we develop this association of ideas the quicker is the response to word hearing.

In conclusion, permit me to predict for the acoustic method that when all children in schools for the deaf have been tested for their acoustic function; when it is found that over 30 per cent of all such children have remnants of hearing that can be utilized and conserved and stimulated to a higher degree of perception; when authorities of instruction have been convinced that the application of the acoustic method will result in untold benefits; when the financial per capita expense of the education of the deaf child will no longer be estimated in figures with a pad and pencil, but rather by the ultimate practical results obtained—then a new era for the deaf will dawn, and a "peace that passeth all understanding" will come into the hearts of all peoples interested in this splendid cause.

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XXXI.

RECENT CLINICAL OBSERVATIONS ON INVOLVEMENT OF THE BLOOD STREAM IN OTITIC DISEASE.*

BY HAROLD I. LILLIE, M. D.,
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There is a difference of opinion regarding the surgical management of phlebitis of the lateral or sigmoid sinus when it is complicated by septicemia from suppurative disease of the temporal bone. Most observers have insisted that the sinus should be ablated and the vein tied in all cases.

Ballance says, "Want of agreement among serious workers about a grave question of surgical treatment arises from lumping together of clinical conditions having an essentially different pathologic basis, or from essential pathologic conditions not being clearly understood. . . . All true and lasting surgical practice is based upon pathology, and when once pathology of an affection is clearly appreciated, divergence of view as to its treatment ought to disappear."

The views that operation on the vein is always required, and that operation is never required, would seem equally erroneous. In this country the geographic section in which the disease is encountered seems to influence the surgical measures used in combating it. It would seem that there is a stage in the disease when eradication of the adjacent infected process would control its further extension and be preferable to subjecting the patient to the greater danger of opening the sinus or tying off the jugular vein. Some of the more conservative aural surgeons have held to this view. My own recent experience in the management of cases of early involvement of the blood stream from suppurative disease of the temporal bone which have been dealt with conservatively, has convinced me that the cases can be handled satisfactorily if they are properly chosen.

*In Contributions to Medical Science, dedicated to Aldred Scott Warthin. George Wahr, 1927, pages 613-624.

The fact that the lateral or sigmoid sinus might become infected from suppurative disease of the temporal bone was not recognized until long after brain abscess and meningitis of otitic origin were known.

Abercrombie reported the condition encountered at necropsy in 1816. Many observers recognized that the treatment of infection of the sigmoid or lateral sinus was surgical; still, Zaufal in 1880 was the first to suggest that the sinus should be opened and the jugular vein tied. It should be borne in mind that the disease, as seen by early observers, was in the advanced stages. The first instance in which operation was performed is credited to Lane, in 1888. Balance's first experience was in 1889. He suggested that the vein be ligated in two places and divided between the ligatures.

At the present time aurists are familiar with aids to diagnosis to which the older observers did not have recourse, and thus a tentative diagnosis may be made much earlier in the disease than formerly. Obviously such an advantage enhances the prognosis.

CLINICAL, PATHOLOGIC CONSIDERATIONS.

Phlebitis of the sigmoid sinus must not be confused with mural, obliterating or degenerating thrombosis. By phlebitis is meant inflammation of the wall of the vessel before mural thrombosis has taken place. In general, it may be said that infection, or the products of infection, of suppurative disease of the temporal bone may extend to the venous sinuses in one of several ways: (1) By direct absorption of the infection through the small vessels in the diseased mastoid, sometimes without visible changes in the appearance of the sinus wall; (2) by the development of phlebitis of the sinus, without parasinus abscess, by continuity from the infected bone of the sinus groove; (3) by direct extension into the sinus because of the breaking down of the wall of the sinus by a parasinus abscess; (4) by the breaking down of the intima of the sinus by the disease process and onset of mural thrombosis; or (5) by the breaking down of obliterating thrombosis within the sinus.

The first two methods of extension are obviously encountered in the earlier cases and are characteristic of the types to be considered here. In the first and second types of the dis-

ease, removal of the primary source of the infection by a complete mastoid operation without interference with the venous sinus or jugular vein offers, in a large measure, sufficient chance for control of the disease, so that it may be chosen as a well directed conservative measure, particularly when the patient is otherwise in good or fair general condition. If subsequent surgical measures become necessary the delay occasioned by the conservative measure will not have materially increased the risk to the patient.

CLINICAL OBSERVATIONS.

From time to time it has been observed that, immediately after the removal of packing from the wound of the mastoid in certain cases in which the sigmoid sinus had been exposed at operation, there may be a sudden high rise in temperature, with or without a chill, followed by an uninterrupted convalescence if there was no further surgical interference. Such a course of events could be explained by the absorption of infected material or the products of infection, into the sinus, as the removal of packing might disturb a natural protective barrier. In certain cases in which the mastoid dressing had not been changed early enough, a sudden high rise in temperature might be relieved by removing the packing and flushing the wound. In cases in which parasinus abscess was encountered and a natural protective barrier had been established there was rarely any febrile reaction after operation. If symptoms and signs of involvement of the sinus were present before mastoid operation but no objective sign of involvement of the wall of the sinus could be made out at the time of the operation, noninterference with the sinus itself was followed by uninterrupted convalescence. This could be explained on the basis of removal of the primary focus of infection before damage to the wall of the sinus had taken place.

These observations led to the hypothesis that in the early cases absorption took place directly into the blood stream through the vasa vasorum into the sinus. It is well known that the cranial sinuses are but sparsely covered with lymphatics, and therefore it is not probable that absorption had taken place through such a channel. In certain cases of parasinus abscess in which a natural barrier had been established absorption was

prevented. In fact, the presence of visible changes over the surface of the sinus furnishes no proof that the wall of the vessel is involved through its entire thickness.

Therefore, it was assumed that in certain of the early cases, even those in which septicemia could be demonstrated by blood culture, the disease might be controlled by the institution of suitable methods so that further surgical interference would be unnecessary. To this end the diseased bone was removed by a complete mastoid operation, the wall of the sinus uncovered until normal appearing dura was encountered and the dura not forced away from the edges of the bone by packing the wound, but instead allowed to tampon against the edge of the bone under intracranial pressure, and thus afford mechanical obstruction to involvement. Recent experience with such cases in the early stage of the disease has justified this procedure.

CLINICAL MATERIAL.

During the last year five cases of acute suppurative otitis media and mastoiditis were observed in which signs and symptoms of invasion of the blood stream suddenly appeared during the course of an otherwise satisfactory convalescence. At no time before the marked change in the course of the disease had there appeared any of the usual positive symptoms or signs of surgical mastoiditis. All but one had been observed from the onset of the otitis media.

ABSTRACTS OF CASE HISTORIES.

Case 1.—A man, aged twenty-seven, presented himself at the clinic October 8, 1925, complaining of stuffiness in the right ear following a cold in the head. Examination disclosed slight injection along the handle of the malleus on the right drum. In the course of forty-eight hours definite suppurative otitis media developed, for which myringotomy was performed. Convalescence was uneventful, and in ten days the ear had ceased discharging and the wound in the drum had closed. Three weeks later, the patient returned complaining of the same symptoms, and the drum was found injected and full. Myringotomy again relieved the symptoms. After another week night pain was complained of; this could not be accounted for except

on the assumption that the infection had extended to the sinus or dura.

The results of the general physical examination were negative, including blood counts and blood cultures. There was no fever. The patient was sent to the hospital for observation, and the following day the temperature rose to 101°. The blood culture was repeated, and pneumococcus, type iii, was found. The leukocytes numbered 8,500. The general physical condition appeared satisfactory, but because of the positive blood culture it was decided to give a blood transfusion by the citrate method, in which gentian violet, 80 cc. of a 0.25 per cent solution, was added to 500 cc. of the blood, because the dye was thought to be specific for this organism.

Transfusion was carried out on the third day after the patient's entrance to the hospital and was followed by an immediate drop in temperature to normal (Fig. 1.). A mastoid operation was performed on the seventh day, and the mastoid disease was found to be of hemorrhagic type. Many cells were filled with thin secretion and a few granulations. The sigmoid groove was not diseased (Fig. 2) but was purposely removed. The outside wall of the sinus was studded with petechial hemorrhages, with some exudate over the surface (Fig. 3). Further exploration was made until more normal dura was encountered, but the sinus was not interfered with. Convalescence was uneventful.

Comment.—If another case of this type was encountered it would be impossible to make an earlier diagnosis, as there were no symptoms to cause one to suspect extension of the infection. Blood transfusion and gentian violet seemed to control the fever, but had no effect on the night pain. The operative findings were sufficient to account for the septicemia and night pain. The patient did not appear very ill at any time. Had he not been kept under close observation, the early involvement of the blood might not have been recognized. None of the signs of surgical mastoiditis was present—that is, there was no sagging of the superior posterior angle, no mastoid tenderness, and very little discharge from the middle ear.

Case 2.—A boy, aged thirteen, presented himself at the clinic May 18, 1926, complaining of a sensation of fullness in the right ear and a discharge of two weeks' duration. Chills

and fever had occurred during the previous two days. The boy looked very ill and had a fever of 104° . The leukocytes numbered 10,400. The differential blood count showed 79 per cent neutrophils. The general physical examination was negative. The blood culture showed hemolytic streptococcus of a virulent type.

The operation on the left mastoid was performed May 19. A small cell mastoid was encountered. No pus was found in any of the cells, but there was a little granulation tissue in the cells around the tympanic antrum. The wall of the sinus was purposely exposed above the knee and was found to be normal. Further exploration down toward the lower turn revealed a definite area of phlebitis which extended to the emissary vein (Fig. 4). The wall of the sinus was changed in character and color, covered with exudate and studded with petechial spots. Apparently mural thrombosis had not taken place; therefore the dura was exposed to the point where it seemed to be normal, but the sinus was not interfered with further.

In the postoperative course, the patient's general condition improved immediately, but after three days there was a rise in temperature to 104° , at which time the wound was dressed and flushed. Following this convalescence was uneventful and the patient was dismissed the ninth day after operation (Fig. 5).

Comment.—That a serious complication had resulted was obvious at the first examination, because the patient appeared very ill. At operation everything was in readiness to tie the vein and ablate the sinus if it seemed necessary. When the type of pathologic change was recognized at operation the more radical operation was deferred. The intentional exposure of the sinus wall with negative findings caused further search for the disease, as it was felt that sufficient cause for the clinical picture would be found somewhere. This is an important obligation and responsibility in the surgical management of mastoid disease.

Case 3.—A boy, aged fourteen, was seen in March, 1926, the fourth day after the appearance of an acute infection in the upper respiratory tract because it was followed by pain in the ears. Examination revealed definite otitis media, for which myringotomy was performed. Following this there was a co-

pious discharge of sanguineous pus. After two weeks the usual improvement did not occur under the routine treatment. The patient was sent to the hospital, and there was a gradual rise in temperature to 102° for three days. The fever was remittent at night. Blood culture revealed a positive hemolytic streptococcus. The leucocytes numbered 13,000; the differential count was practically normal.

On the nineteenth day of the disease, mastoid operation was performed, and a small cell type of mastoid involved by the hemorrhagic type of disease was found. The sinus was purposely exposed, although the sinus plate showed no signs of disease and definite phlebitis was encountered. The sinus wall was exposed to normal appearing dura without further interference. Following the operation there was slight fever, but the patient showed every sign of doing well and the convalescence was uneventful.

Comment.—Something about the condition of the patient suggested that he was not doing well, even in the face of rather negative clinical findings. The gradual rise in the height of the remittent type of temperature and the finally positive blood culture were sufficient to warrant exploration in spite of negative clinical signs. Again, a small cell mastoid, without any breaking down of cells or destruction of the sinus plate, was found. The area of phlebitis was found immediately on uncovering the sinus and it was very extensive. The postoperative course was satisfactory, as the patient was infinitely improved within a day after the operation (Fig. 6).

Case 4.—A boy, aged thirteen, was seen February 27, 1926, with an acute infection of the upper respiratory tract, and acute suppurative otitis media with fever of 103° . The general physical examination was negative. Because he appeared ill and nonresistant to infection, observation in the hospital was advised, but the parents refused it until four or five days later. Careful examination then revealed nothing other than the otitis to account for the condition. The blood culture showed hemolytic streptococcus, and on the second day operation was performed. The mastoid was found to be of the diploetic type and the disease of definitely hemorrhagic character. The sinus lay well back, and the sinus groove was found extensively involved but not broken down. The sinus wall was exposed and found

covered with a greenish secretion and areas of phlebitis and granulations extending below and posterior to the sigmoid sinus and involving also the emissary vein. This was uncovered to the normal dura and no further interference with the sinus was undertaken.

The condition of the patient remained fair for three days after the operation and the temperature reached nearly normal limits. Then for three days there was a low grade remittent type of fever. Transfusion of 500 cc. of blood, together with 80 cc. of a 0.25 per cent solution of gentian violet, was given. Neither parent was a suitable donor. There was little change in the temperature for two days and then it remained at a more or less normal level (Fig. 7).

Comment.—This patient had never been robust. It was felt that he should be carefully observed from the onset, but for financial reasons he was not put in the hospital. Definite signs of mastoiditis were lacking, but the positive blood culture and appearance of the patient made operation imperative. The involvement of the sinus was more extensive than in the previous cases but the type of disease was essentially the same.

The postoperative course was none too satisfactory and further operation seemed required. It was decided to use blood transfusion with gentian violet as a supportive measure. The clinical improvement was so decided that further interference became unnecessary. The clinical course in this case leads me to suspect that the wall of the vein was involved in its entire thickness, as this would account for the slower return to normal.

Case 5.—A boy, aged five, was seen March 1, 1926, because of pain in the ear during the course of infection in the upper respiratory tract. The examination revealed suppurative otitis media, for which myringotomy was performed. There was also acute follicular tonsillitis. The patient was sent to the hospital and was found to have a high fever with slight remissions, the type which might be accounted for by the tonsillitis. Blood cultures showed hemolytic streptococcus. On the sixth day of the disease the tonsillitis had practically subsided and a mastoid operation was performed. Operation revealed a small cell mastoid with pus present in all cells and welling up under pressure from below. A little behind the sig-

moid sinus, extensive necrosis had taken place, and the sinus was found covered with a large parasinus abscess. The wall of the dura was much thickened and covered with what appeared to be healthy granulations. A slight visible pulsation suggested mural thrombosis. As the lesion was thoroughly revealed, normal looking dura was found. The sinus filled well and nothing further was done. The operation had very little effect on the course of the temperature (Fig. 8). Three days later a transfusion of 250 cc. of blood with 2 mg. of mercurochrome for each kilogram of body weight was given as a supportive measure and because it was thought that mercurochrome might be a specific for this organism. However, this had little effect on the clinical course, and in six days the sinus was ablated and the vein tied. The postoperative temperature course was stormy, but the child seemed to be improving daily and the final outcome was satisfactory.

Comment.—It might be argued that too much dependence was placed on conservative measures. In another such case I would ablate the sinus and tie the vein at the first operation. It will be noticed that this case was different from the others in that great destruction of the mastoid took place in a short time. Instead of the hemorrhagic type of disease, it was definitely suppurative, although the same type of organism was present as in cases 2, 3 and 4.

Although the amount of granulation over the sinus is not a true index to the involvement, still the fact that there was visible pulsation, which accompanies mural or obliterating thrombosis, should have been sufficient reason for opening the sinus. The rapid course of the disease was another factor. Earlier interference did not seem justifiable on account of the tonsillitis. Blood transfusion and mercurochrome had a supportive effect but not so pronounced as in the other case.

This case was actually not suitable for conservative measures. The delay in ablating the sinus and tying the vein added some risk, although the final result was satisfactory.

DISCUSSION OF CASES.

The cases were encountered over a period of six months and not in an epidemic. In cases 1, 2, 3 and 4 the clinical course, the type of arrangement of the mastoid cells, the type of dis-

case, and the effect of the operation were much the same, although the organism in cases 2, 3 and 4 was the hemolytic streptococcus, while in case 1 it was pneumococcus, type iii. Usually more pain is expected in the small cell mastoid than was noted in this group. The pain may be explained on a mechanical basis.

The great advantage of laboratory aids to diagnosis stands out. The blood culture is far more important than the leukocyte or differential count. Laboratory tests were not made to be of use in statistics, but only when necessary for diagnosis. In young patients it is so difficult to carry on the necessary postoperative dressings that other discomforts are best avoided.

CONCLUSIONS.

1. Whether the sinus need be ablated and the jugular vein tied in cases with infection in the blood stream originating in the temporal bone at the time of the original mastoid operation will depend on the type of disease, the physical condition of the patient and the judgment of the aural surgeon.

2. In certain cases with early involvement of the blood stream removal of the diseased bone and sufficient exposure of the sinus wall will control the disease.

3. Little real harm to the patient can result from conservative measures if cases are well chosen.

4. Blood culture is the most important laboratory aid to diagnosis.

5. Blood transfusion with the addition of gentian violet or mercurochrome is beneficial. At least a supportive effect and perhaps a definite therapeutic effect may be expected.

6. The general appearance of the patient following operation is a fairly satisfactory index of the physical condition, and this will obviate annoying the patient with unnecessary laboratory tests.

7. All cases of acute otitis media should be closely observed.

8. Early mastoid operation is not indicated in all cases of otitis media without definite physical evidence of surgical mastoiditis, but is demanded in cases in which there is evidence of extension of the infection through the mastoid to the blood stream.

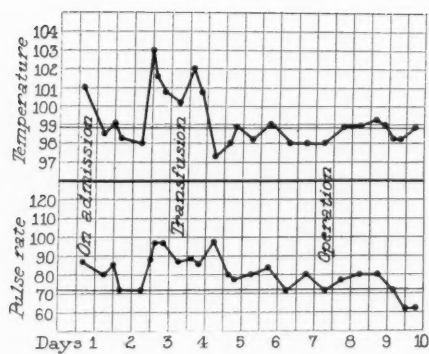


Fig. 1 (Case 1).

Striking effect on temperature curve of transfusing blood and gentian violet.

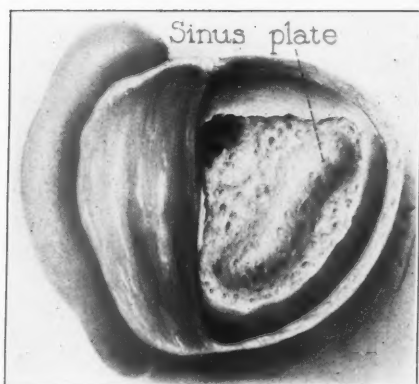


Fig. 2 (Case 1).

Purposeful exposure of sigmoid sinus plate without apparent disease.



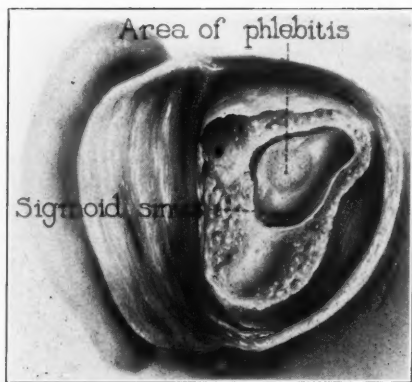


Fig. 3 (Case 1).

Purposeful exposure of wall of upper turn of sigmoid sinus disclosing area of phlebitis.

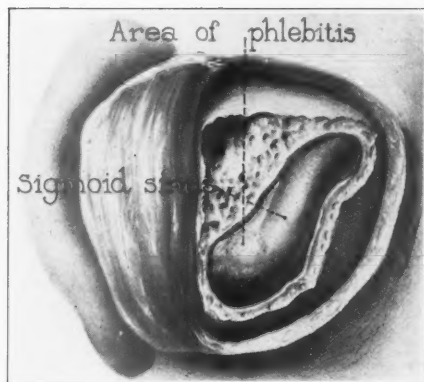


Fig. 4 (Case 2).

Exposure of upper turn of sigmoid sinus with negative findings.
Exposure toward lower turn revealing area of phlebitis.

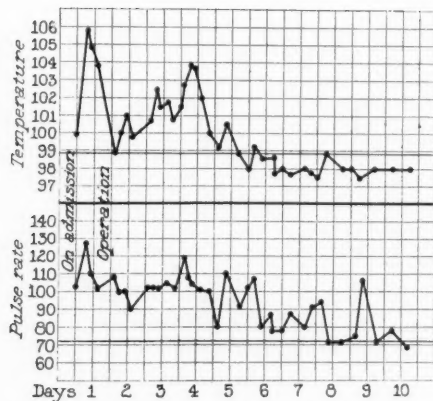


Fig. 5 (Case 2).

Effect of dressing and flushing wound on postoperative febrile reaction.

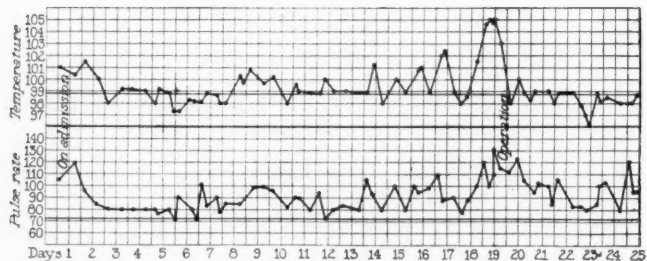


Fig. 6 (Case 3).

Sudden change in temperature curve at time of involvement of blood stream. Effect of operative interference on the temperature curve manifest.

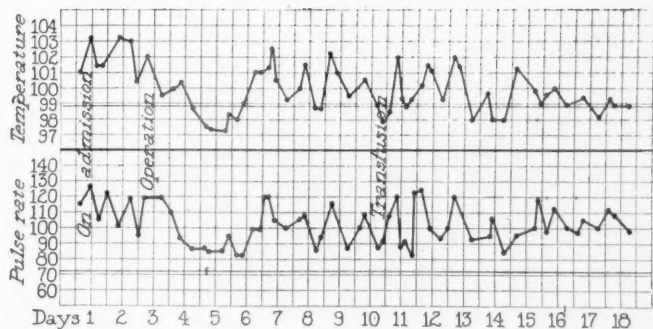


Fig. 7 (Case 4).

Little apparent effect of operation or transfusion on temperature curve in spite of continual improvement in general appearance of patient.

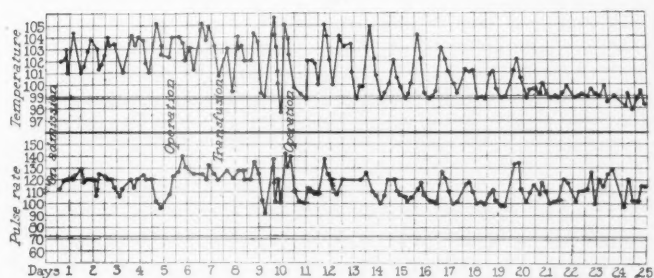


Fig. 8 (Case 5).

Slight effect of operations and transfusion on temperature curve. No objective signs of improvement for several days after second operation.

XXXII.

AN ANALYSIS OF THE REPORTS OF FATALITIES
FOLLOWING THE USE OF LOCAL ANESTHET-
ICS, WITH A RÉSUMÉ OF RECENT STUDIES
ON THE SUBJECT, INCLUDING A SUM-
MARY OF FOURTEEN ADDITIONAL
FATALITIES NOT HITHERTO
RECORDED.

By EMIL MAYER, M. D.,

CHAIRMAN OF THE PERMANENT COMMITTEE ON TOXICITIES
FOLLOWING LOCAL ANESTHESIA OF THE AMERICAN
MEDICAL ASSOCIATION,

NEW YORK.

EVENTS LEADING TO THE REPORT OF THE COMMITTEE.

By unanimous vote of the Section on Laryngology, Otology and Rhinology of the American Medical Association, at its meeting held in 1919, the chairman of the section, Dr. L. W. Dean, was authorized to appoint a committee to study and report on the advantages and disadvantages of local anesthesia.

The subject was introduced by the presentation of a letter from Dr. Torald Sollmann, chairman of the Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the association, requesting the appointment of such a committee, offering to aid the committee both financially and otherwise.

Dr. Emil Mayer, New York City, Dr. Ross Hall Skillern, Philadelphia, and Dr. Robert Sonnenschein, Chicago, were appointed and agreed to serve.

The chairman of the committee promptly drafted a plan and scope of the investigation, which was strictly adhered to.

The first report of the committee was read the following year at the meeting of the section. It included the results of a questionnaire sent to a limited number of otolaryngologists, animal investigations specially made for the committee and

was followed by discussion thereon by men who had made special study on local anesthesia.

In addition to the study of the difference between local and general anesthesia, the committee reported the brief histories of eighteen deaths not previously recorded in medical journals. The full report was published.¹

In the belief that a fuller investigation would result in the discovery of more deaths, all of which would form the basis of a better knowledge of the subject, the committee recommended a further study of the subject in which the cooperation of every member of the section on oto-rhinolaryngology was to be secured. This was agreed to, and the same committee appointed with the addition of the then secretary of the section, Dr. Wm. B. Chamberlin of Cleveland. This committee was enabled to report twenty-two additional deaths among otolaryngologists,² none of them previously recorded.

The committee further agreed that the question of toxicity had never before been fully studied in any collective investigation, that it was important to study it from other points of view and that all sections of the association interested should participate in this further study with a view to adding to our knowledge of the subject, ascertaining the causes of fatalities and assisting in the prevention of fatalities as far as possible.

This recommendation was sent to the chairman of the Council on Pharmacy and Chemistry of the association, received his endorsement and that of the Board of Trustees of the association, the latter agreeing to defray all necessary expenditures.

Realizing the importance of the appointment of representative men from each specialty who would be willing to serve, who were unbiased and who were competent in studying details and forming deductions, Dr. Sollmann, as chairman of the Council, appointed a nominating committee who would confer with the chairmen of the eight sections interested and present the names of those chosen for a collective investigation. The nominating committee consisted of Dr. Reid Hunt, Harvard University Medical School; Dr. Robert A. Hatcher, Cornell Medical College, and the writer. The latter was asked to serve as chairman of the new committee. The following were then nominated in due course:

MEMBERS OF THE COLLECTIVE INVESTIGATION.

Representing Surgery, Dr. Elliott C. Cutler, now of the Lakeside Hospital, Cleveland; Medicine, Dr. David I. Macht, Johns Hopkins University, Baltimore; Pharmacology, Dr. Robert A. Hatcher, Cornell Medical College, New York; Pathology, Dr. Chas. Norris, Chief Medical Examiner for New York City, Bellevue Hospital; Stomatology, Dr. Henry S. Dunning, New York City; Genito-urinary Surgery, Dr. Alexander Randall, Philadelphia; Ophthalmology, Dr. Robert S. Lamb, Washington, D. C.; and Laryngology, Dr. Emil Mayer, New York City.

As before, a distinct plan and scope of the investigation was prepared by the chairman and a questionnaire sent to all members of the association registered in each section represented.

Each member of the committee received the replies of the questionnaire sent to his section, who sent all replies to the chairman of the committee.

Thus the chairman was able to collect and analyze reports of forty-three new and hitherto unpublished fatalities by local anesthetics. All data in connection therewith were gathered and sent to each member previous to the meeting of the entire committee at the New York Academy of Medicine, on October 8, 1923.

MEETING OF THE COMMITTEE.

Every phase of the subject was given careful consideration, and the discussion showed that every member present had studied the data submitted by the chairman.³ The committee was unanimous in all its conclusions and of the opinion that the main objectives sought had been attained. At the end of the meeting each member in turn expressed gratification and enthusiasm at being permitted to participate in a scientific study of such magnitude, the first collective investigation that had ever been made on this subject, and the hope was expressed that the publication of its report would result in great benefit to the profession and to humanity. To this each added a tribute to the chairman of the committee, which was most highly prized by the latter, and which modestly forbids the writer from repeating.

The voluminous report was referred to the executive committee for condensation and presentation in proper form as the conclusions of the committee. Their report was then sent to each member of the committee, endorsed by each and was then sent to the chairman of the Therapeutic Research Committee. The latter went over the report with care and expressed his appreciation of the work, with special mention of the leadership of the chairman, drafted a résumé thereof, which was signed by each member of the Therapeutic Research Committee and published in the Journal of the American Medical Association as a special article in an introductory paragraph.

The chairman had tabulated the reports of forty-three deaths but for want of space this was not published. It is herewith appended.

In addition to the publication of all the reports, these were read at meetings of the section and special societies, where free discussion was possible and comments invited. At none of these was there any dissent. The research was commended and special mention of gratitude to the Therapeutic Research Committee of the Council on Pharmacy and Chemistry was expressed for instigating the investigation.

RECEPTION OF THE REPORTS.

The reception of the reports by the profession at large has been remarkable and practically unanimous. Not only in personal communications but in many letters from far and near. From Hong Kong, Australia, Alberta in Canada, as well as from many parts of the United States; prominent medical journals have commended editorially thereon most favorably.

The program committee of the American Academy of Ophthalmology and Otolaryngology requested a review of this subject by the chairman for its meeting in Montreal, Canada, in 1924.⁴ Individual members of the profession felt themselves impelled to present their own experience in the use of local anesthetics. Dr. James J. King⁵ detailed his own experience in local anesthesia in nose, throat and ear surgery, publishing, as a second part of his valuable treatise, the complete reports of all the committees heretofore mentioned. This publication has since proved of value to those wishing to study the subject, as the large supply of reprints generously given to the

committee by the courtesy of the Journal of the American Medical Association had become entirely exhausted. These reports so collected are of great convenience to students of the subject who would otherwise have to search through original sources of publication.

THE RECOMMENDATION OF THE COMMITTEE.

One operator accustomed to the use of cocain paste, made by saturating cocain crystals with epinephrin solution, objected to the declaration that it should not be used.⁶ He declares that he uses as much as from four to six grains of cocain crystals mixed with epinephrin, always using epinephrin first, in one nasal operation, and never has he seen the slightest evidence of toxicity. Moreover, he states that he often uses that concentration at the same time that he uses a general anesthetic. The paste, he states, is not suitable for all purposes. He has *never ventured* to use it in the pharynx.

COMMENT.

This subject of the use of cocain paste received careful consideration by our committee. It had been claimed for this concentration that absorption could not occur, as it produced such powerful constriction of the blood vessels and thus prevented absorption.

We presented altogether three deaths following the use of this method and felt that the argument in its favor was proven false. In this present report another fatality is recorded following the use of cocain paste.

It was felt by us that all therapeutic rules hitherto taught were that the minimum concentration that would produce the desired effect should always be given, and in view of the number of fatalities, unusually large, considering the comparatively infrequent use of cocain, we believed this method to be dangerous. It may here be stated that if the amount of cocain used were within the bounds of safety recommended by the committee, it made no difference if it were applied as a paste or not. Thus one colleague states⁷ that he uses this paste method, carefully weighs the amount to be used, again weighing any remainder, and thus secures complete local anesthesia, using only 65 milli-

grams at each operation; we believe that he is well within the line of safety.

The writer of this article doubts whether any American otolaryngologist would ever use as much as four or six grains of cocain at one sitting, as his English colleague does, nor can he conceive of the necessity of using a local and a general anesthetic at the same time.

Dr. Macht⁸ made a series of experiments in the laboratory of the Johns Hopkins Medical School on the effect of local anesthetics in general anesthesia and concludes that to give a general anesthetic on top of a local anesthetic would be a very dangerous procedure. This he says is pharmacologically plausible on account of the depressant effect on the medulla or heart or both. Eggleston and Hatcher⁹ found that cats anesthetized with hydrated chloral succumb to doses of procain that are harmless to normal animals. Finally, the defendant of the paste method confesses to a fear of cocain when he says he has *never ventured* to use it in the pharynx. We may well support him here, for we report in this present series a fatality following the use of cocain paste in a tonsil operation.

One more point may be considered in defense of the statement that cocain paste should not be used, and that is, throughout our investigation we found always a tendency among surgeons to use far larger doses of local anesthetics than recommended by the manufacturers. This was demonstrated, for instance, in the Butyn deaths.

DANGERS OF INTRANASAL APPLICATION AND INJECTION.

It was also demonstrated to the committee that death may follow the intranasal application of a local anesthetic; also that injection submucously meant rapid absorption.

COMMUNICATIONS.

The great number of letters received were commendatory. The interest in the subject is still acute. Although so many years have elapsed since the publication of our collective investigation, four letters were received during the past month relative to the subject from different parts of our country. One writer recommends that the subject be taken up in each State association. He writes appreciatively of the help he has

had from a study of the reports, wants to pass it along and see it passed to others.

PREVENTION OF TOXICOSIS.

Tatum, Atkinson and Collins presented¹⁰ a study of the use of barbitol to prevent cocain poisoning. Leshure¹¹ and Williams¹² have endorsed this procedure. King¹³ advises the use of magnesium sulphate with novocain and morphin. E. F. Howard¹⁴ conducted an investigation among otolaryngologists in Mississippi. He states that "we have been decidedly reckless in the use of cocain in intranasal operation, and it is so entirely unnecessary. Synergistic analgesia, novocain injections and nerve blocking will do all that one need, or if a man cares to rely on cocain, the preliminary use of adrenalin, plus a high concentration of cocain *in limited quantities* will bring him well within the margin of safety. It is the quantity of cocain absorbed that constitutes the danger, and since I have been accurately estimating the amount I used, now over two years, I find that I can perform a simple resection of the septum with an average of approximately 65 milligrams, and this in cases where no synergist is employed. I have a measured 15 drops of solution of adrenalin and a weighed 100 mgr. of cocain put on a tray, and I never fail to send back part of the latter to be weighed in the laboratory."

Professor Gustav Alexander,¹⁵ in his recent visit to this country, stated to the writer that cocain is not permitted in his clinic or in his office.

NEW LOCAL ANESTHETICS.

In a recent publication in the Wiener Med. Wochenschrift,¹⁶ the query as to advances in local anesthesia is thus answered:

"There is great danger in the administration of cocain, discovered by Koller in 1884, as it is especially toxic. An application of a 15 per cent solution may readily paralyze the respiratory center, hence its use should be extremely limited. Its active principle has been shown to be a benzoyl, hence there are a number of preparations to take its place, such as orthoform, anesthesin, novocain (procain), stovain and tropacocain. None of these can entirely replace cocain, as they do not induce surface anesthesia. Hence the search for some drug

to replace the poisonous cocain is justifiable. We have been enabled to produce synthetic cocain from well known chemicals. Among these we have psicain, which is in every way a substitute for cocain. It is much less poisonous, decomposes rapidly, produces surface anesthesia. It seems to the referees that psicain will be the cocain of the future. There is another preparation not yet marketed called ekcain. Novocain is principally used because it contracts blood vessels. Stovain produces hyperaemia. The effect of novocain may be increased by adding an alkaline solution. A similar increase of the effect of novocain follows the addition of epinephrin. Anemia follows with resultant increase of the anesthesia. A further increase of the anesthesia is induced by adding sulphate of potassium to the novocain adrenalin solution." (Prinz¹⁷ has made use of this combination of sulphate of potassium with novocain and adrenalin, following the suggestion originally made by Kochmann, Hoffmann and Zorn, for over six years in the Evan's Dental Institute of the University of Pennsylvania and has published his formula more than two years ago. He had then averaged 15,000 injections of novocain per year for the past five years, using over a pound of novocain yearly.

In addition Prinz has tested a great many of the readymade solutions of novocain offered to the profession, such as carpules, ampules, etc., and he has invariably found that the epinephrin is decomposed. He favors the preparation of a standard solution of procain (novocain) with sodium chloride and potassium sulphate. To this solution the epinephrin is added, but under no circumstances should the solution with epinephrin be boiled and never more than five minims used at one time. Alkali free glassware should be used. The standard solution consists of: Sodii chlorid, C. P. 7.0; potass. sulph., C. P. 4.0; A. Dist., 1000 cc. 400 cc. of this solution should be boiled with 6.0 procain in an Erlenmayer flask, 4 to 5 minutes. Filter when cool. When needed, 10 cc. of this procain solution is mixed, not boiled, with 4 or 5 drops of epinephrin solution. No matter how much procain solution is used, five drops of the epinephrin solution is the maximum quantity permissible.

The writer's attention has been directed to a new local anesthetic, the notes of which have been shown to him. It is not

known if it has been submitted to the Council on Pharmacy and Chemistry for endorsement. "It was discovered by a French chemist, August Chesnais, and is called *NiKetol*. It is said to be a synthetic product of organic origin, made by the association of two ethers, each of which shows strong anesthetic properties. The one is a derivative of the para-amino benzoic series, and the other an ethylic ether of phthalamic acid. *NiKetol* is a stable salt, which keeps indefinitely in isotonic solutions, is slightly soluble in water, more soluble in alcohol, easily dissolved in physiologic serum and almost insoluble in ether. *NiKetol* solutions are sterilized in an autoclave at 120° C., without any modification of its composition or properties. It produces immediate, powerful and lasting local anesthesia, is slightly constrictive and obviates the addition of epinephrin in most cases. When applied there is an absence of causticity, producing no eschars or necrosis.

For surface application ampoules of 10 cc. or 20 cc. in 5 per cent concentrations are pink in color, the 10 per cent are blue. The solution is practically nontoxic, as 150 cc. have been administered without discomfort. Its effect is immediate; healing is not delayed. It may be applied with a swab or as a spray.

To prevent spread of infection in an inflamed tissue injection should be made from the periphery to the center and a new needle used for every injection."

NEW FATALITIES.

With no attempt at inquiry, the writer has secured data of fourteen deaths occurring in 1925, 1926 and 1927. These followed immediately upon the use of a local anesthetic for various diseased conditions. The local anesthetics used and the number of fatalities are:

Cocain, 3; cocain-novocain (reported as novocain only), 2; novocain, 8; butyn, 1.

COCAIN DEATHS.

1. For tonsillectomy, June 23, 1925. Female, aged 22. Application of a freshly prepared solution of cocain hydrochlorid 4 per cent. Became very excitable, followed by general intermittent convulsions. Throat cleansed, morphin and atropin

injected, followed by epinephrin and artificial respiration. Death in 45 minutes.

2. For tonsillectomy, May 10, 1927. Female, aged 17. Morphine, 1/6 grain, before operation; 2 cc. of a 5 per cent solution of magnesium sulphate injected one hour before; 2½ grains powdered cocaine moistened with epinephrin solution swabbed on tonsils. Death promptly ensued. No necropsy—status lymphaticus given as cause of death.

3. Following accidental injury to axilla, August 10, 1927. Female, aged 9. Four cc. of a 1 per cent solution of cocaine were injected about the wound. Convulsions and respiratory failure followed in a few minutes. Necropsy showed markedly enlarged thymus weighing 58 grs.

COCAINE AND NOVOCAIN DEATHS.

1. For tonsillectomy, September 23, 1925. Female, aged 35. Cocaine solution, 10 per cent, swabbed on throat; amount not stated; then 5 drachms ⅛ per cent solution of novocain were injected. Death immediate, attributed to novocain. Necropsy showed no pathologic changes.

2. For tonsillectomy, May 10, 1927. Female, aged 17. (Statement of operator: Injection of morphine sulph., ¼ gr., with atropine sulph., 1/150 gr., one-half hour before operation. Ten per cent cocaine solution applied, 4 or 5 drops, to anterior and posterior pillars of tonsils. Similar application of adrenalin, 1 to 1,000, then injection of ½ per cent novocain and adrenalin, three injections.) About 2 cc. in all. Both tonsils removed. Sudden cyanosis and collapse. Stimulation and artificial respiration to no avail. Death attributed to novocain (sudden).

Necropsy showed enlarged thymus, 30 grs. No cocaine was found chemically in liver.

NOVOCAIN DEATHS.

1. For removal of tonsils, September 8, 1925. Female, aged 20. Six cc. of a ½ per cent solution of novocain injected. One tonsil removed. Sudden edema and collapse. Death promptly followed. No necropsy.

2. For prolapse of rectum, October 27, 1925. Male, aged 61. Spinal anesthesia. Amount of novocain used not stated.

3. For hemorrhoids, August 7, 1926. Male, aged 42. Morphine, gr. 1/6, previously injected; 12 cc. of 1/2 per cent solution of novocain with epinephrin injected. Convulsions and death.

4. Duodenal ulcer; pyloric obstruction, July 23, 1927. Colored female, age 43. Peritoneal cavity opened; 5 cc. of a 0.5 per cent solution of novocain injected. Convulsions and death. Necropsy, thymus present weight about 12 grs. (persistent thymus). No novocain or cocain found in liver chemically.

5. For prostatectomy, September 30, 1927. Male, aged 51. Adenomatous hypertrophy, prostate gland. Supra pubic cystotomy performed under novocain infiltration. Two weeks later perineal prostatectomy under sacral and para-sacral anesthesia; novocain injected; amount not stated. Convulsions and death. Necropsy showed chronic myocarditis.

6. Tumor right parotid, October 15, 1927. Male, aged 45. Tumor had been extensively radiated and was very painful, about the size of an olive. Patient was very apprehensive. Forty cc. of a 1 per cent solution of novocain were injected. Patient died suddenly while tumor was being removed. Necropsy showed thymus and thyroid somewhat enlarged. Brain examined for novocain and cocain negative.

7. Hernia, bilateral, inguinal, December 6, 1927. Male, aged 69. Fecal vomiting; obstipation 3 days; vomiting 2 days. Auricular fibrillation, myocarditis.

Novocain, 1 per cent, for skin; 1/2 per cent for deep infiltration. Died on operating table.

8. For tonsillectomy, December 2, 1927. Male, aged 26. Except for chronic persistent eczema both hands, for some months, was entirely well. Evidence of definite chronic infection of tonsils. Seated, without previous administration of any narcotic nor hypodermic injections, the throat was injected at four points with approximately 2 1/2 cc. of a 0.5 per cent solution of novocain made two days previously, to which had been added just before injection six drops of epinephrin solution to one ounce of novocain solution. Immediate nausea. Operation begun on right tonsil; patient gagged and vomited. Tonsil forceps removed. Convulsions, extreme cyanosis, marked dilatation of pupils. Violent convulsions with clonic muscular contractions that threw him practically out of the

chair. Recumbent position; artificial respiration, oxygen; 1 cc. epinephrin, 1/1,000, injected directly into heart muscle. Convulsions lasted 30 seconds. Death immediate. No necropsy. Skiagraph records no evidence of thymic widening. Contents of fluid remaining in syringe, also original vial of solutions sent to State toxicologist for examination. Both solutions found to be of same strength, contained novocain hydrochloride; no cocain found. Novocain, 2.79 grs. to fluid ounce of solution. In view of the careful examination preoperative of patient and the small dose of novocain, the operator feels that this was a case of anaphylactic poisoning.

BUTYN DEATH.

1. For tonsil operation, September 1, 1925. Female, aged 20. Butyn injected $\frac{1}{2}$ per cent (states that not more than 2 cc. were used). Convulsions occurred in three minutes, lasted five minutes; became unconscious; removed to hospital. Operation began at 12:35 p. m.; died at 1:20 p. m. Necropsy: Thymus large, fleshy (no weight given).

SUMMARY.

For some unknown reason, procain* is still charged with being the cause of a fatality when admittedly cocain had been used immediately prior to the procain, in both instances 10 per cent cocain was applied. The number of cocain deaths should, therefore, be stated as five, over 40 per cent of the number recorded. It is difficult to understand why cocain is used in such concentrations for tonsil operations and the warnings against the use of both drugs unheeded. The proportion of fatalities is exceedingly large when we consider the relative infrequency in which cocain is used as compared to procain. It is the writer's opinion that the proportion of procain used to cocain must be many thousand of the former to one of the latter. At least two of these cases were practically moribund before operation. We note particularly the many dentists who use procain without a single fatality. None has

*Some confusion exists because of the use of the term procain for novocain. The writer has used the term novocain in the cases reported, as the impression might arise that the fatalities were due to an American made preparation.

any cocain in his office, as a rule. The number of procain deaths is very small, considering the vast amount of it used. In view of the large number of cocain deaths again recorded and the fact that it is conceded to be a poisonous drug by the majority of the medical profession, the writer earnestly recommends the cessation of the use of this drug except for examination purposes or endolaryngeal operations, before an accident occurs that would assuredly result in punitive damages, compelling such caution out of self interest if not for the protection of the individual.

Of interest in the series is the death following the use of cocain paste in the pharynx, which has already been considered in the body of this paper. The death from butyn in such small an amount and concentration, even though associated with an enlarged thymus, is most noteworthy and indicates that it may act as a virulent poison, especially dangerous with such complication.

As thirteen of these deaths occurred in a limited though very populous area, we have no means of knowing whether there has been an increase or decrease of these accidents since our last report. It is noteworthy that not one *single instance* of substitution is recorded in this group, though it was quite frequent in the former. The custom still persists that these deaths are said to be due to status lymphaticus, a definition of which would be most acceptable. That certain individuals are highly susceptible to local anesthetics there can be no doubt, nor is it to be inferred that the operating surgeon might have foreseen the presence of that condition. The fact that fatalities have occurred in the hands of competent and experienced operators is undoubted. Cocain in high percentages is undoubtedly the most dangerous. Our reports have dealt only with the fatalities, with no mention of the many cases of temporary conditions following the use of local anesthetics, and their publication has stimulated further research.

The appointment of a permanent committee has shown its value in that many questions relative to local anesthesia have been sent to the chairman and these have been invariably answered. The publication of the names of those who have taken part in the investigation is done to indicate the thoroughness

of the study and the independent standing of the individual members of the committee.

All new local anesthetics begin well, as a rule. Time and experience only will tell how safe they are. At present procain seems to be the most reliable and safe among those in general use.

RECOMMENDATIONS.

The addition of potassium sulphate to the novocain solution, as recommended by Prinz, is advisable.

It would be better to explain to the patient that there would be slight pain with the first introduction of the hypodermic needle and none thereafter, thus obviating the use of cocain. The danger of substitution should always be borne in mind. As a matter of great value to future investigators, the chemical examination of the anesthetic used in fatalities should always be undertaken. Solutions sometimes become concentrated.¹⁸ The more frequent publication of fatalities is advisable. That local anesthesia has come to stay is undoubted.

The writer wishes to express his gratitude to many colleagues for kindly endorsement, cooperation and encouragement, to the capable aid of his associates in the various committees, and acknowledges with thanks the courtesy of Dr. J. J. King in permitting a copy of the summary herewith published from his valuable book. (See chart inside back cover.)

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666 WEST END AVENUE.

The Semi-Centennial Meeting of the American Laryngological Association will be held in the Hotel Raleigh, Washington, D. C., on May 1st, 2nd and 3rd, 1928. The guest of honor will be Sir St. Clair Thomson of London, England, who is an Honorary Fellow of the Association. A number of papers will be devoted to the achievements of Laryngology during the last half century, both in civil life and in the Army and Navy during the World's War. All members of the profession interested in Laryngology are cordially invited to be present.

The American Board of Otolaryngology will hold an examination in Minneapolis on Monday, June 11th, during the meeting of the American Medical Association.

Those wishing to submit applications for this examination are requested to communicate with the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Bldg., Omaha, as soon as possible.

H. P. MOSHER, President

On January 7th, a dinner was given for General Herbert S. Burkitt of Montreal, at which time the medal of honor given by the Academy was presented to him, together with the certificate of honorary membership.

The speech of presentation was made by Dr. Ross Hall Skillern, of Philadelphia, president of the Academy, 1926-1927.

TABULATION OF THE FORTY-THREE LOCAL AN

No.	Date of death	Record number	Reported to doctor	Sex	Age	Place of death	Drug used	Strength	Amount	How applied
1	1915	CCCI	Randall	M	50	Unknown	Alypin	4 per cent	2 or 3 dr.	Injected in urethra
2	1920	CCCH	Randall	M	28	Cocaine	2 per cent	Injected in urethra
3	1918	CCCHH	Randall	Negro M	Hospital	Cocaine	2 per cent	2 dr.	Injected in urethra
4	N. S.	CCCIV	Randall	M	23	Cocaine	N. S.
5	1920	CCCV	Randall	M	62	Cocaine	4 per cent	10 c.c.	Injected in urethra
6	1920	CCCVI	Randall	M	55	Cocaine	½ per cent	Further information ref. Dr.
7	1919	CDI	Norris	F	25	Dispensary	Cocaine and procaine	N. S.	Applied and injected latter
8	1920	CDII	Norris	Negro M	35	Hospital	Cocaine	10 per cent	Injected in urethra
9	1920	CDIII	Norris	F	24	Office	Cocaine Adrenalin Procaine	20 per cent 10-15 dr. 1 per cent	Swabbed and injected
10	1920	CDIV	Norris	F	24	Dispensary	Cocaine	1/10 per cent	Injected
11	1921	CDV	Norris	M	23	Office	Atropine Morphine Cocaine Adrenalin	½ gr. in ½ hour N. S. N. S.	Injected Spray Injected
12	1921	CDVI	Norris	M	47	Hospital	Cocaine Procaine	10 per cent ¼ per cent	100 minims	Applied Injected
13	1921	CDVII	Norris	Negro M	29	Dispensary	Morphine Atropine Cocaine Procaine	¼ gr. 1/150 gr. 10 per cent 0.5 per cent	¼ gr. 3 dr.	Injected Swabbed 3 times Injected
14	1921	CDVIII	Norris	M	22	Dispensary	Cocaine Procaine Adrenalin	10 per cent 0.5 per cent 5 minims	3 dr.	Spray Injected
15	1922	CDIX	Norris	F	37	Office	Cocaine Adrenalin	10 per cent 1/10 per ct.	Swabbed and injected
16	1921	CDX	Norris	M	34	Dispensary	Morphine Cocaine Procaine	⅛ gr. 10 per cent ½ per cent	4 dr.	Swabbed and injected
17	1922	CDXI	Norris	F	23	Office	Atropine Morphine Cocaine Adrenalin	1/150 gr. ¼ gr. 4 per cent 1-2000	Injected Spray Packing
18	1923	CDXII	Norris	F	32	Office	Cocaine	100 per cent	4 per cent applied then cocaine paste
19	1922	CDXIII	Norris	M	53	Hospital	Stovaine	Spinal puncture 4-5 lumbar
20	1921	CCI	Mayer	M	20	Hospital	Cocaine Procaine Adrenalin	20 per cent 1 per cent 8 drops to 1 oz.	15-20 minims ½ oz.	Swabbed Injected
21	1921	CCII	Mayer	N. S.	N. S.	Office	Cocaine	2 per cent	2 cc. or more	Injected
22	1918	CCIII	Mayer	F	20	Hospital	Cocaine Procaine Adrenalin	10 per cent 1 per cent	3 dr.	Swabbed Injected
23	1923	CCIV	Mayer	F	38	Hospital	Butyn Cocaine Morphine	¼ per cent 4 per cent ⅛ gr.	2 dr.	Injection Applied Injected
24	1923	CCV	Mayer	F	35	Office	Butyn Morphine Atropine	5 per cent ⅛ 1-200 gr.	N. S.	Swabbed and packed Injected
25	1923	CCVI	Mayer	M	16	Office	Butyn Morphine Atropine	5 per cent ⅛ gr. 1-200 gr.	N. S.	Swabbed and packed Injected
26	1923	CCVII	Mayer	M	26	Office	Butyn	Injected
27	1923	CCVIII	Mayer	M	70	Hospital	Butyn	Less than 1 per cent; 5 tablets in	40 c.c.	Spinal injection sacral foramen

LOCAL ANESTHETIC DEATHS PREVIOUSLY REPORTED. ARRANGED BY DR. EMIL MAYER

How applied	Condition for which drug was used	Position of patient	Symptoms preceding death	How soon after using drug did death occur	Measures used to resuscitate	History of epilepsy	Cause of death as reported	Autopsy	Remarks by attending physician or member of Committee
Injected in urethra	Urethral stricture	Lying down	Convulsions; marked cyanosis	Five to seven minutes	N. S.	N. S.	Alypin poison	Yes	Multiple abscesses liver and spleen found.
Injected in urethra	Cystoscopy	Lying down	Convulsions	One minute	Adrenalin injection attempted	No	Cocaine poison	No	Previous cystoscopy two days before caused wound and rapid absorption.
Injected in urethra	Stricture	Lying down	Coma	Within a few hours	N. S.	N. S.	Cocaine poison	No	Previous attempts to dilate. Stricture parts badly torn and bleeding.
	Stricture	Lying down	Shock; unconscious	Rapid	N. S.	N. S.	Cocaine poison	Yes	General hyperemia of the viscera. Persistent thymus.
Injected in urethra	Cystoscopy	Lying down	Violent cough; unconscious	Immediately	Adrenalin and pulmotor	No	Cocaine poison	No	Had bronchiectasis and interstitial nephritis.
Hemorrhoids. Information refused to both Dr. Randall and Dr. Mayer									
Applied and injected latter	Nose operation	Sitting	Hysterical; breathing lessened	At once	Pulmotor; oxygen; strychnia	No	Cocaine procaine death	Yes	Status lymphaticus.
Injected in urethra	Stricture	Lying down						Yes	Liver examined chemically, cocaine not found.
Abbed and injected	Tonsil operation proposed	Sitting	Convulsions and cyanosis	Ten minutes	Artif. resp.; dilated sphincter ani; adrenal inj. over heart; pulmotor	No	Cocaine poison	Yes	Status lymphaticus. Chemical examination, liver and kidney. Cocaine not found.
Injected	Tonsil operation completed	Sitting	Convulsions	About one-half hour		No	Cocaine poison	Yes	Acute hemorrhagic pancreatitis. Status lymphaticus extreme congestion all organs; liver examined chemically, negative.
Injected	Tonsil operation proposed	Sitting	Collapse; stertorous resp.; coma	Over six and one-half hours	Artificial resp.; oxygen; digitalin; colonic irrigation	No	Cocaine	Yes	Congestion all organs. No cocaine found in liver. Coma.
Ray injected	Tonsil operation in part	Sitting	Cyanosis; convulsions	One-half hour	Ether; morphine; camphor; caffeine; adrenalin	No	Cocaine	Yes	Chr. interstitial myocarditis; marked coronary sclerosis. Congestion all organs.
Injected abbed 3 times	Tonsil operation proposed	Sitting	Convulsions	Twenty-five minutes	Inhal. ammonia; digitalin; camphor; atropine; strych.	No	Cocaine	Yes	All organs congested.
Ray injected	Tonsil operation proposed	Sitting	Convulsions	Three minutes	Oxygen; morphine; dilat. sphincter	No	Cocaine	Yes	Status lymphaticus. Persistent thymus; general visceral congestion.
Abbed and injected	Tonsil operation proposed	Sitting	Severe pain in head; convulsions	Few minutes	Pulmotor two hours	No	Cocaine	No	Fright.
Abbed and injected	Tonsil operation begun	Sitting	Cyanosis	Few minutes	Oxygen; strychnine	No	Cocaine	Yes	Status lymphaticus; extreme congestion all organs.
Injected	Nose operation completed	Sitting	Convulsions	Two hours	Arom. spir. am.; oxygen; strych.; morph. ¼ gr.	No	Cocaine	Yes	Status lymphaticus. Shock (½ gr. morphine—one-half hour). E. M.
Over cent aped then cocaine paste	Puncture antrum	Sitting	Stertorous breathing, frothing at mouth	Twenty minutes	Artif. resp.; whiskey	No	Cocaine	Yes	Ac. pulmonary congestion and edema. General visceral congestion. Suppurative frontal sinusitis.
Anal puncture lumbar	Scrotal hernia	Lying down	Inability to breathe	Shortly after returning to recovery room		No	Stovaine in a syphilitic	No	Paralysis of phrenics.
Abbed	Tonsil op.—right removed, while removing left symptoms showed	Sitting	Closed jaw, pallor; tremor lower limbs; clonic spasms	Five minutes	Artif. resp.	Yes	Cerebral embolism	No	Epileptic (?).
Injected	Tonsil operation proposed	Sitting	Respiratory failure	One hour	Artif. resp.	No		Yes	Eight months previously had similar injection for extraction of teeth with no symptoms, hence no idiosyncrasy.
Abbed injected	Tonsil operation completed	Sitting	Respiratory failure	Six minutes	Pulmotor	No		Yes	Enlarged thymus found.
Injection	Tonsil operation intended	Sitting	Faintness; convulsions	Few minutes	Atropine; ether; strych.; artif. resp.	No	Paralysis respiratory and circulatory centers	Yes	Patient died before operation was attempted. Approves small preliminary injection. Cardiac hypertrophy
Abbed and injected	Septum resection intended	Sitting	Convulsions	Fifteen minutes	Artif. resp.; many stimulants	No	Purulent ethmoiditis	No	Probably release of intracranial pus.
Abbed and injected	Resection; operation intended		Convulsions; cyanosis	Fifteen to twenty minutes	Many drugs, massage of heart	No	Disturbance endocrine system	Yes	Idiosyncrasy.
Injected	Tonsil operation intended	Sitting	Fright; faintness; convulsions				Surgical shock	No	
Anal injection foramen	Prostatectomy intended	Lying down	Convulsions; twitching eyes; collapse	Five to seven minutes	Artificial resp.; epinephrin	No		No	Spinal fluid withdrawn post mortem and examined for butyryl negative

23	1923	CCIV	Mayer	F	38	Hospital	Butyn Cocaine Morphine	$\frac{3}{4}$ per cent 4 per cent $\frac{1}{2}$ gr.	2 dr.	Injection Applied Injected
24	1923	CCV	Mayer	F	35	Office	Butyn Morphine Atropine	5 per cent $\frac{1}{2}$ 1-200 gr.	N. S.	Swabbed a n d packed Injected
25	1923	CCVI	Mayer	M	16	Office	Butyn Morphine Atropine	5 per cent $\frac{1}{2}$ gr. 1-200 gr.	N. S.	Swabbed a n d packed Injected
26	1923	CCVII	Mayer	M	26	Office	Butyn	-----	-----	Injected
27	1923	CCVIII	Mayer	M	70	Hospital	Butyn	Less than 1 per cent; 5 tablets in 100 c.c. not d i s s o l v e d entirely	40 c.c.	Spinal injection sacral foramen
28	1921	DI	Dunning	M	55	Office	Unknown	Unknown	N. S.	Injected
29	1922	DII.	Dunning	M	42	Office	Cocaine Adrenalin	Flakes 1 to 1000	-----	3 applications
30	1922	CL	Macht	M	33	Hospital	Procaine Adrenalin	$\frac{1}{2}$ per cent	1 oz. 10 minims	Infiltration
31	1921	CLI	Macht	M	3	Hospital	Procaine	1-200 gr.	-----	Infiltration
32	1921	CLII	Macht	F	39	Hospital	Cocaine Substitu- tion	15 per cent	-----	Infiltration
33	1922	LXI	Cutler	M	26	Hospital	Apothesine a n d a d- renalin	2 per cent	$\frac{1}{2}$ oz.	Injected in ton- sil only
34	1919	LXII	Cutler	F	17	Hospital	Morphine Atropine Cocaine Adrenalin	$\frac{1}{4}$ gr. 1/150 gr. 10 per cent $\frac{1}{2}$ c.c. of 1-1000	-----	----- Swabbing $\frac{1}{2}$ c.c. injected each side
35	1921	LXIII	Cutler	F	26	Hospital	Morphine Atropine Cocaine Procaine	$\frac{1}{4}$ gr. 1/150 gr. 20 per cent $\frac{1}{2}$ per cent	5 cc.	Swabbing a n d injected
36	1921	LXIV	Cutler	F	Not given	Hospital	Apothesine	$\frac{1}{2}$ per cent	$5\frac{1}{2}$ oz.	Injected
37	1921	LXVII	Cutler	M	31	Dispensary	Cocaine Procaine	10 per cent $\frac{1}{2}$ per cent	-----	Swabbing Injected
38	1922	LXVIII	Cutler	F	41	Hospital	C o c a i n e a n d a d- renalin	1/10 per ct. 1 min.-1 dr.	$\frac{1}{4}$ gr.	Injected
39	1918	LXIX	Cutler	F	36	Hospital	Cocaine	$\frac{1}{2}$ per cent	20 cc.	Injected Infiltration
40	1921	LXX	Cutler	M	22	Hospital	C o c a i n e a n d a d- renalin Procaine	10 per cent $\frac{1}{2}$ per cent	8 cc.	Swabbing Injected
41	1921	LXXI	Cutler	F	16	Hospital	Apothesine Adrenalin	1 per cent	$1\frac{1}{4}$ oz.	Injected Infiltration
42	1922	LXXII	Cutler	M	25	Hospital	Procaine Adrenalin	$\frac{1}{2}$ per cent	200 cc.	Injected
43	1921	LXXIII	Cutler	F	61	Hospital	A p o t h e- sine with c h l o r e- tone Adrenalin	$1\frac{1}{2}$ per cent $\frac{1}{2}$ per cent	2 cc. N. S.	Spinal injection

(King, J. J., Local Anesthesia in Otolaryngology and Rhinology.
Paul B. Hoeber, Inc., New York, 1926.)

tion ed ted	Tonsil operation intended	Sitting	Faintness; convul- sions	Few minutes	Atropine; ether; strych.; artif. resp.	No	Paralysis respi- ratory and cir- culatory centers	Yes	Patient died before opera- tion was attempted. Ap- proves small preliminary injection. Cardiac hyper- trophy
bed a n d ed ted	Septum resec- tion intended	Sitting	Convulsions	Fifteen min- utes	Artif. resp.; many stimulants	No	Purulent eth- moiditis	No	Probably release of intra- cranial pus.
bed a n d ed ted	Resection; oper- ation intend- ed	Convulsions; cya- nosis	Fifteen to twenty minutes	Many drugs, mas- sage of heart	No	Disturbance en- docrine system	Yes	Idiosyncrasy.
ted	Tonsil operation intended	Sitting	Fright; faintness; convulsions	Surgical shock	No	
l injection al foramen	Prostatectomy intended	Lying down	Convulsions; twitch- ing eyes; collapse	Five to seven minutes	Artificial resp.; epinephrin	No	No	Spinal fluid withdrawn post mortem and examined for butyn; negative.
ted	Tooth extrac- tion	Sitting	Respiratory failure	At once	Digitalin; brandy	N. S.	No	Relative stated that patient had some heart disturb- ance; this was not con- firmed.
ications	Chr. disease of antrum	Sitting	Collapse; coma at once until death	Forty hours	Artificial resp.; arom. spir. am- mon.; brandy	Cocaine primary cause	Yes	Chronic interstitial neph- ritis; status lymphaticus.
ration	Tonsil operation	Reclining on table	Sick at stomach; no convulsions	At once	Pulmotor and heart stimulants	No	Status lymphat- icus	Yes	Previous examination for enlarged thymus negative.
ration	Sloughing of skin from burn	Reclining on table	Convulsions; cya- nosis	Immediately after injection	Artificial resp.; oxygen	No	Burns of body and extremities	No	No.
ration	Resection of rib	Reclining on table	Convulsions; cya- nosis	Immediately after injection	Artificial resp.	No	Cocaine poison; myocarditis; peri- and endo- carditis	Yes	Mistake of using 15 per cent cocaine for the procaine intended. Fluid injected was examined afterward showing cocaine.
ted in ton- nly	Tonsil operation	Reclining on table	Cried out; fell to floor; clonic spasms	Twenty-five to thirty min- utes	Pulmotor and stim- ulants; ether in- jected and morph. atrop.	No	None given	No	Tablets 221 Apoth. B. sent to Mfrs. for analysis; re- port all right.
.....	Tonsil operation	N. S.	Operation complet- ed; sent to room; found in collapse	Ten minutes	N. S.	No	None given	No	Cocaine—idiosyncrasy.
bing injected side	Tonsil operation	Unknown	Faintness; convul- sions; cyanosis	Four hours	Stimulants; mor- phine; intravenous saline	No	No	Cocaine?
ted	Thyroidectomy	Recumbent	Convulsions; cyano- sis; faintness	Thirty min- utes	Artificial resp.	No	No	Not a very toxic thyroid from description. Evident poisoning by apothesine (E. C. C.).
bing ted	Tonsil operation	Sitting	Convulsions; respi- ratory and cardiac failure	Four minutes	No	Status lymphat- icus	Yes	Study of autopsy protocol does not show status lym- phaticus. Idiosyncrasy (E. C. C.).
ted	Tonsil operation	Sitting	Fainted; slow and difficult resp.	One and one- fourth hours	Artificial resp.; stimulants; pul- motor	No	Pulmonary em- bolus	No	No evidence of embolus to reporter.
ted ration	Skin graft	On back	Severe convulsions	Less than five minutes	No	Acute cocaine poisoning	No	Used by mistake.
bing	Tonsil operation	Sitting	Faint convulsions	After twenty minutes	Morphine and ether injected and in- haled	No	Cocaine and ad- renalin poison- ing	No	Idiosyncrasy.
ted	Enterostomy for ileus	Lying down	Felt as if she was be- ing put to sleep; eclamptic conv.	Five to ten minutes	No	No	Cannot explain this unless there was a mistake in the drug which the doctor denies (5-5 grs. apothesine E. M.).
ted ration	Thoracoplasty for tuberculosis	Horizontal	Profound cardiac weakness	Forty-two hours	Stimulations of all kinds	No	Operator thinks procaine respon.	No	Shock is my impression (E. C. C.).
l injection	Exploratory lap- arotomy	Flat	Dyspnea severe; pal- lor; shock; air hun- ger	Forty-five minutes	Cardiac asthma	Resp. paral. due to spinal anes- thesia	No	Medullary paralysis.

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